VOLUME LIV MAY, 1944 NUMBER 5

THE LARYNGOSCOPE

FOUNDED IN 1896
BY
MAX A. GOLDSTEIN, M. D.

PUBLISHED BY
THE LARYNGOSCOPE
640 SOUTH KINGSHIGHWAY ST. LOUIS (10), MO., U. S. A.

NOTICE TO CONTRIBUTORS

THE LARYNGOSCOPE reserves the right of exclusive publication of all articles submitted. This does not preclude their publication in Transactions of the various Societies.

Manuscripts should be typewritten, double spaced, on one side of paper only and with sufficient margins to allow for corrections.

References should be complete: authors surname, initials, title of article, journal, volume, page, month, year.

Six illustrations will be furnished for each article without cost to author. Authors will please limit illustrations to six or assume the expense of additional illustrations.

Proofs will be submitted to authors for corrections. If these are not returned, articles will be published as corrected in this office:

Reprints will be furnished at the following prices:

WITHOUT COVER

	250 Copies	500 Copies	1000 Copies	2000 Copies
Four Pages	$ 5.75	$ 7.00	$ 9.50	$14.50
Eight Pages	12.00	14.50	19.50	29.50
Twelve Pages	17.00	21.25	30.00	47.50
Sixteen Pages	21.50	26.50	36.50	56.50
Twenty Pages	26.25	32.75	46.00	73.50
Twenty-four Pages	30.50	38.00	53.00	83.00
Thirty-two Pages	40.50	48.25	65.00	98.50

WITH COVER

Four Pages	$ 9.75	$12.50	$18.00	$29.00
Eight Pages	16.00	20.00	28.00	44.00
Twelve Pages	21.00	26.75	38.50	62.00
Sixteen Pages	25.50	32.00	45.00	71.00
Twenty Pages	30.25	38.25	54.50	87.00
Twenty-four Pages	34.50	43.50	61.50	97.50
Thirty-two Pages	44.50	53.75	73.50	113.00

Express charges to be paid by consignee.

THE
LARYNGOSCOPE.

VOL. LIV MAY, 1944. No. 5

THE USE OF SULFA DRUGS IN THE MASTOIDECTOMY WOUND WHERE COMPLETE ENCLOSURE IS EMPLOYED.*

ARTHUR J. HERZIG, M.D., New York.

When it is realized that 40 years ago the average time to heal a mastoid wound was from two to four months, it seems sort of miraculous that we now heal them by first intention, prevent unsightly scars and depression of the mastoid wound. While mastoid wounds have been healed by first intention with blood clot, etc., there always was that fear that we were bottling up some infection which might form an extradural abscess, etc. Today our modern chemotherapy to all intents and purposes makes the procedure foolproof.

There is nothing original in closing a mastoidectomy wound completely as Blake,[1] of Harvard, reported a series of cases in which he employed primary suture some 40 years ago, using the blood clot as a medium for filling the exenterated wound. The author has employed closing the wound completely, using blood clot (1913) ; 1-200 acriviolet in the wound before closing same in three cases (1931) and seven cases in which he has filled the wound with equal parts of sulfanilamide and sulfathiazole powder. The author has been suturing the periosteum separately with catgut since 1913. In all of the cases so sutured, with drain or without, there has been no wound depression.

---

*Read at the Meeting of the New York Academy of Medicine, Section on Otolaryngology, New York, Nov. 17, 1943.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Dec. 6, 1943.

1906 — The blood clot dressing as suggested by Dr. Blake and Dr. Reik[2] is prepared in the following manner: "After thorough exenteration of the mastoid cavity the same is thoroughly dried and then permitted to fill with blood from the surrounding soft tissues. A complete sutural closure with continuous subcutaneous silver wire is the best. These wounds heal in from five to 10 days."

March, 1906 — Henry Reik — Blood clot dressing first recommended for use in the mastoid operation by Dr. Clarence J. Blake, of Boston. After the wound is filled with blood, the clot tends to organize and form new tissue similar to that enclosing the clot. The fibrous framework of the clot forms the scaffolding on which the new tissue is built. Fresh granulations spring from the walls of the cavity and grow out into the clot, forming a new fibrous connective tissue. Osteoblasts are sent out from the bony walls or periosteum to convert the fibrous substance into bony tissue. This process only extends inward from the outer walls for a short distance, while the center of the new formed tissue remains fibrous. Nielchinkoff, Nuttall, Fedor, etc., state that the normal blood possesses bacteriocidal powers, varying in degree in its antagonism to different micro-organisms. The microbe-destroying power is in the blood serum. It has been determined that blood serum removed from the body acts more rapidly and energetically than the plasma and lymph in the body. The nucleins appear to be the proteid that is secreted by the leucocytes that has this bacteriocidal power. The clotted blood appears to have greater bacteriocidal power than the circulating blood, as the leucocytes break down rapidly and give up the protein (nuciein). Nuclein acts only in an alkaline medium; if alcohol is added, the function of the nuclein is completely destroyed. Excessive alkalinity or bichloride also destroy the action of the nuclein.

September, 1906 — Sprague[3] first conceived idea in 1892. Completely closed mastoid wounds, using no drains; blood clot filling mastoid cavity, in 16 cases. Average healing time seven to 15 days.

September, 1906 — William Sohier Bryant[4] follows the

procedure of placing a small cigarette drain in the operated wound for 24 hours. After removal the cavity is filled with blood serum, after which the wound closes by first intention. He has followed this procedure in simple cases; also those with epidural and episinus infections.

June, 1908 — Samuel McCullagh,[5] before mastoidectomy, incises the membrana tympani freely and irrigates the ear with a solution of bichloride of mercury followed by saline solution. After the removal of the bone the cavity is flushed with normal saline solution and sponged dry. A running subcutaneous suture is then introduced. A piece of rubber tissue drain is introduced between the lowest two stitches to drain same. After 24 hours the drain is removed and kept out unless the wound has become infected, in which case the wound is dried out and the drain reintroduced.

1913 — Holmes[6] — "Blake reported his results of Blood Clot Dressing in 1891 before the American Otological Society." After mastoidectomy Holmes fills the wound three times with H_2O_2; dried and then filled with alcohol for three minutes, after which the wound is thoroughly dried. Arterial rather than venous blood is used. Periosteum and skin are then sutured, no drains being used. Blood clot, however, fills the external auditory canal. When the drum heals a disagreeable odor ensues. Of the cases reported, seven were acute mastoids and 33 were radicals. In acute and chronic cases with partial breaking down of the clot, the upper three-quarters of the scar always held perfectly. Perfect healing in 41 per cent of the cases and almost perfect in 41 per cent means 82 per cent of practical results is sufficient justification for a continuation of this method.

1919 — Davis[7] — After exenteration of the mastoid cavity, incised the membrana tympani thoroughly, establishing free communication between the middle ear and mastoid cavity. cleansed area, packed with iodoform gauze, closed wound completely except for a small opening at the lower angle through which one end of the gauze was protruded. In 24 hours the packing was withdrawn, allowing the blood to fill the cavity. The lower angle of the wound was closed with

adhesive plaster, with the removal of the sutures on the second or third day. Davis flushes ear to antrum and return with 3 per cent iodine, then warm alcohol, lastly with warm physiologic saline solution.

1920 — Coates[8] quotes Reik's paper, "The Ideal Mastoid Operation" (1916) before the American Otological Society, where he reaffirmed all his views as expressed at that time. Reik favors the complete blood clot and takes issue with those who, like Coates, prefer in many cases to use a modification. Coates flushes the wound with hot salt solution; no chemical sterilation is employed. After this, artery clamps and retractors are removed, and the wound allowed to fill with blood, the skin incision being closed by a subcutaneous silver wire suture, etc. Eighty per cent cures reported.

1941 — Livingston[9] advocates closure of mastoidectomy wounds using sulfanilamide.

December, 1942 — Wilbur D. Currier[10] states the use of sulfathiazole in snugly closed mastoidectomy wounds without drainage accelerates healing and decreases hospitalization.

December, 1942 — Tucker and Flake[11] reported observations on a series of 46 patients. Group A — 16 patients — Complete mastoidectomy performed; wound completely closed without drainage; sulfadiazine powder poured in. Group B — 15 patients — Mastoidectomy wounds were closed without drainage, sulfadiazine being administered orally only. Group C — 15 patients had a simple mastoidectomy operation performed; these wounds being simply drained, no sulfadiazine being placed in the wound or given orally. *Comment:* Hospitalization — Groups A and B — 10 days. One patient in Group B had wound breakdown. In Group C the average hospitalization stay was 21 days. The above quoted authors did not state whether their patients were adults or children.

April, 1943 — Ralph A. Fenton[12] — "In almost every case during the past 18 months we have found it possible after mastoidectomy to make primary closure of the wound by means of careful hemostasis and dusting in of sufficient sulfathiazole powder to cover the walls of the operative cavity. It is undesirable to fill the cavity, for an excess of the powder

may act as a foreign body in a large granulating wound. Gauze packings or rubber drains are not used."

If I have overlooked any additional prior reports, I wish to apologize to the authors for so doing.

Procedure: Mastoid cavity must be thoroughly exenterated. Care must be taken that no debris, such as pieces of bone, torn periosteum or blood clot are left. No chemical or saline solution should be used to flush the wound. The wound is simply dried with gauze after all bleeding points have been controlled. The cavity must be dry. Equal parts of sulfanilamide and sulfathiazole powder are then packed into the mastoid cavity, including the antrum. More combined powder is dusted on the closed periosteum. The skin is then closed with silkworm gut or Michel clips; more of the combined powder is then dusted on the dried, closed wound. Some combined powder is placed in the external auditory canal before the usual wick is introduced. The regulation mastoid dressing is then applied. The skin sutures or clips are left for five days. After their removal there is no necessity for further dressings.

Case 1: M. R., female, age 5 months, admitted Feb. 21, 1942. Previous history — for the past 24 hours there had been pain in the right ear with redness and edema in the region of the right mastoid bone. For past two weeks had been troubled with a head cold. Upon examination the eardrum was found to be injected with a loss of the light reflex. Pharynx and nasal chambers injected. X-ray revealed small infantile mastoids showing no evidence of bone destruction. Feb. 21, 1942 — Hemoglobin, 75 per cent, 11.8 gm. per 100 cc.; R. B. C., 3,800,000; W. B. C., 25,000; neutrophiles, 89 per cent, of which 76 per cent were mature and 14 per cent immature; lymphocytes, 20 per cent. Blood Laughlin negative. Report of mastoid culture taken at the time of operation — hemolytic streptococcus. March 2, 1942 — Hemoglobin, 65 per cent, 10.2 gm. per 100 cc.; R. B. C., 4,000,000; W. B. C., 10,600; neutrophiles, 45 per cent, of which 37 per cent were mature and 8 percent immature; lymphocytes, 58 per cent; eosinophiles, 2; mononuclears, 5; anisocytosis moderate; polychromatophilia very slight; achromia moderate; microcytes rare; macrocytes rare. March 3, 1942 — Blood culture negative. Urinalysis negative throughout. Operative findings: After the usual preoperative preparation, a curvilinear incision was made and a large amount of pus escaped (subperiosteal abscess). There was a large perforation in the cortex over the antral region. The mastoid cell (antrum) was soft and readily curetted. There was a large amount of pus throughout the remainder of the mastoid. Sulfanilamide powder was placed in the wound and the periosteum and skin completely closed. Dry dressing applied. Patient discharged March 6, 1942.

Case 2: H. G., male, age 10 months, admitted Oct. 15, 1942. Following a head cold, the right mastoid bone presented a large amount of postauricular swelling. A. P. O. M. present; incised. Bulging of superior quadrant

only. Cervical adenitis on right side. Persistent high temperature for two days prior to admission. Oct. 15, 1942: Hemoglobin, 62 per cent, 9.8 gm. per 100 cc.; R. B. C., 3,590,000; W. B. C., 21,400; mature neutrophiles, 52 per cent; immature, 7 per cent; lymphocytes, 41 per cent. Operation under ether anesthesia. A curvilinear incision was made in back of the right ear. After the periosteum was elevated, the mastoid antrum was entered. This child had an unusually low dural plate. The tip cell was fairly well developed and full of pus. Culture was taken. After cleansing the wound thoroughly, sulfathiazole powder was poured into the wound, completely filling same. Three catgut sutures approximated the periosteum and Michel clips completely closed the skin. No drain inserted. Dry dressing applied. Culture from mastoid cavity — Streptococcus viridans. Urinalysis negative. Oct. 21, 1942 — Hemoglobin, 63 per cent, 9.9 gm. per 100 cc.; R. B. C., 4,000,000; W. B. C., 12,400; mature neutrophiles, 36 per cent; immature, 2 per cent; lymphocytes, 60 per cent. Patient discharged Oct. 29, 1942.

Case 3: N. R., female, age 4 years, admitted March 4, 1943. For the past three weeks left A. P. O. M. present; large amount of discharge, the perforation being in the superior-posterior portion of the tympanic membrane. Marked swelling present over the mastoid bone with considerable amount of pitting upon pressure. Diagnosis: Subperiosteal abscess with anterior and cervical adenitis present. X-ray revealed profuse clouding showing evidence of mastoiditis. March 5, 1943 — Hemoglobin, 75 per cent, 11.8 gm. per 100 cc.; R. B. C., 4,500,000; W. B. C., 14,700; mature neutrophiles, 72 per cent; immature, 10 per cent; eosinophiles, 1 per cent; lymphocytes, 15 per cent; mononuclears, 2. After usual preoperative procedure, incision was made one-quarter inch parallel to the pinna, through skin, subcutaneous tissue and periosteum. The periosteum was then elevated, mastoid tip freed of strands from sternocleidal mastoid muscle. A perforation was observed in the cortex about 1 cm. above McEwen's triangle. Pus appeared upon removing the cortex. Culture taken. Mastoid bone was freed of all infected areas and wound dried and packed with sulfathiazole powder. Periosteum approximated with catgut; more of the sulfathiazole powder sprinkled on same and the skin wound closed with Michel clips. No drain inserted. Dry dressing. Culture from mastoid pus — streptococcus hemolyticus. Urinalysis negative. March 10, 1942 — Hemoglobin 78 per cent; R. B. C., 4,800,000; W. B. C., 9,200; mature neutrophiles, 68 per cent; immature, 12 per cent; eosinophiles, 0; lymphocytes, 18 per cent; mononuclear, 1. Patient discharged March 12, 1943.

Case 4: L. H., colored, female, age 12 years, admitted April 1, 1943. Had a profusely discharging left ear for past few weeks. Nocturnal pain has been present at times only. April 1, 1943 — Hemoglobin, 80 per cent; color index, 0.9; R. B. C., 4,670,000; W. B. C., 9,900; mature neutrophiles, 68 per cent; immature, 2 per cent; eosinophiles, 1 per cent; basophiles, 1 per cent; lymphocytes, 25 per cent; mononuclears, 3. April 2, 1943 — X-ray showing clouding of mastoid cells in the region of the periantral triangle, especially marked on the left side. Mastoidectomy performed. After the usual preoperative preparation, a curvilinear incision was made posterior to the auricle and the periosteum elevated. The periosteum was very adherent to the lower half of the mastoid bone and more than double in thickness. After the tip of the mastoid had been freed of all fibres, parallel grooves were made in the mastoid bone starting at McEwen's triangle and including the tip. With curettes and rongeurs the entire tip as well as the mastiod cavity was thoroughly exenterated. Tip cell was full of pus. Culture taken. Small cells were full of degenerated epithelium, polypoid in character. The mastoid antrum was then entered and free bleeding occurred. No exposure of dura or sinus. Wound was filled with equal parts of sulfanilamide and sulfathiazole powder. Periosteum sutured completely with catgut and skin closed with Michel

clips. No drain introduced. Some sulfanilamide powder was dusted into the auditory canal and a small piece of gauze introduced, as is customary. Sterile dressing applied. Forty-eight-hour culture from ear showed hemolytic streptococcus. Seventy-two-hour culture of pus from mastoid wound showed hemolytic streptococcus. April 8, 1943 — Hemoglobin, 79 per cent; color index, 0.9; R. B. C., 4,120,000; W. B. C., 5,400; mature neutrophiles, 54 per cent; immature, 1 per cent; eosinophiles, 2 per cent; basophiles, 1 per cent; lymphocytes, 32 per cent; mononuclears, 10. It is interesting to note that the organism from the preoperative discharge was a hemolyzing staphylococcus aureus while that from the mastoid pus was a hemolyzing streptococcus.

Case 5: A. H., male, age 48 years, admitted April 8, 1943. Three days previous to admission, patient developed an acute pharyngitis, had several hours of chilly sensations and general fatigue. Twenty-four hours later the left ear began to pain. Sensation was of a stinging, painful variety and 24 hours later he heard something "pop," at which time a yellow discharge appeared from the left canal. Pain in the mastoid bone was present from the beginning and was continuous. Patient gave a history of a similar condition 35 years ago. Upon examination the left canal was found to be filled with a thick, creamy, yellow discharge with sagging of the posterior-superior wall. There was exquisite tenderness over the three cardinal points on the mastoid bone. Night sweats were profuse. Pharynx was acutely inflamed. Examinations of the sputum and chest were negative. Stereoscopic examination of the chest was made and no radiographic evidence of pulmonary tuberculosis was observed. X-ray of the mastoids showed clouding with evidence of mastoiditis on the left side with sclerosis in the region of the periantral triangle on both sides. April 8, 1943 — Hemoglobin, 94 per cent, 14.7 gm. per 100 cc.; R. B. C., 5,050,000; W. B. C., 16,000; mature neutrophiles, 69 per cent; immature, 9 per cent; lymphocytes, 19 per cent; mononuclears, 3 per cent. April 9, 1943 — Culture from ear — staph. albus — strep. viridans — 24-hour culture — many Gram positive cocci and diplococci. Culture from mastoid — strep. viridans — 12-hour culture — rare Gram positive diplococci. April 19, 1943 — Hemoglobin, 86 per cent, 13.4 gm. per 100 c. R. B. C., 4,150,000; W. B. C., 9,200; mature neutrophiles, 58 per cent; immature, 8 per cent; eosinophiles, 1 per cent; basophiles, 1 per cent; lymphocytes, 24 per cent; mononuclears, 8 per cent. April 20, 1943 — Smear and culture from mastoid wound — 24-hour culture — no growth; 48-hour culture — occasional colony Gram positive diplococci; final report—hemolytic strep. April 24, 1943 — Hemoglobin, 94 per cent, 14.7 gm. per 100 cc.; R. B. C., 4,970,000; W. B. C., 2,900; mature neutrophiles, 70 per cent; immature, 3 per cent; eosinophiles, 1 per cent; lymphocytes, 25 per cent; mononuclears, 1 per cent. April 9, 1943 — Mastoidectomy, left. Customary curvilinear postauricular incision was made. Bleeding was profuse (B. P., 90/55). After clamping all vessels, periosteum was incised. This was adherent throughout and the fasicular attachment of the sternocleidomastoideus was adherent as far as McEwen's triangle. The temporal ridge, instead of running anteroposteriorly, ran from Henle's spine upward and backward and the thickness was about three times the normal. The mastoid antrum was entered at the usual place and pus under pressure appeared. Parallel grooves were then made to the mastoid tip, which was then removed. The tip had a small cell, full of pus. (Culture taken.) The mastoid bone was of the narrow, elongated type with the lateral sinus far forward. It was the diploic type. In carefully cleaning out the cell the lateral sinus was uncovered. This opening was later enlarged to about one-half inch in length. The vein was covered with unhealthy granulations but these were not disturbed. The mastoid antrum was enlarged and the wound filled with sulfathiazole and sulfanilamide powders, about 1½ drams of each. The periosteum was approximated

with No. 1 catgut sutures; more powder applied and the skin united with silkwormgut. No drain inserted. The external auditory canal was cleansed and a wick introduced as is customary. Dressing applied. Three days following operation a fistula appeared at about the middle of the mastoid wound. Culture was taken of the thick mucoid secretion which was expressed, and the report showed no growth. Fistula was reopened eight days later and final report of culture showed hemolytic streptococcus. Inspection of the mastoid wound about two weeks later showed same to be normally healed, the auditory canal absolutely dry and no perforation remained in the membrana tympana. This case is a very satisfactory one in spite of the fistula and the fact that the patient suffered from malnutrition as well as chronic tonsillitis. It is interesting to note that the culture taken at the time of the operation was predominantly viridans, while that taken from the second opening of the fistula was strep. hemolyticus. Patient discharged April 16, 1943, 17 days after operation.

Case 6: V. H., female, age 15 years, admitted April 15, 1943, complaining of earache. The day previous to admission the patient came to the clinic, where the left drum membrane was incised. She was also suffering from an infected lower bicuspid (Vincent's infection). Examination showed a discharging left ear; no sagging observed. Fluid in the ear was pulsating and mastoid was extremely tender. X-ray examination of both mastoids showed a diffuse clouding of the mastoid cells on the left side, revealing presence of mastoiditis. No radiographic evidence of bone or cellular destruction observed. April 16, 1943 — Mastoidectomy, left. Curvilinear incision made posterior to the auricle. Periosteum elevated; not adherent. Long, narrow mastoid, infantile type. Group of tip cells rather than one single cell present. Lateral sinus far forward. Antrum entered, free bleeding, tip removed; no pus found in antrum or tip cells. Marked necrosis of cell partitions; degenerated membrane in all. Culture taken from blood bathing cells. Note: Preoperative pain in this case was most severe.) After complete exenteration resulting in a smooth cavity, the periosteum was sutured, leaving a small opening through which the cavity was filled with acri-violet solution 1-200 and the sutures tied. Skin approximated with eight black silk sutures. No drain. Usual mastoid dressing. April 15, 1943 — Hemoglobin, 85 per cent, 13.3 gm. per 100 cc.; R. B. C., 4,900,000; W. B. C., 16,400; neutrophiles, 72 per cent; mature, 62 per cent; immature, 10 per cent; basophiles, 3 per cent; lymphocytes, 23 per cent; mononuclears, 2 per cent. April 16, 1943 — Culture of discharge from ear — 12-hour culture — no growth; 24-hour culture — Gram positive diplococci — Gram positive bacilli; three-day culture — Corynebacterium Hodgkinii — hemolytic strep.; four-day culture — no growth. Culture from mastoid wound — no growth after 20 hours. April 23, 1943 — Hemoglobin, 82 per cent, 12.8 gm. per 100 cc.; R. B. C., 4,540,000; W. B. C., 19,950; mature neutrophiles, 67 per cent; immature, 3 per cent; eosinophiles, 5 per cent; basophiles, 1 per cent; lymphocytes, 23 per cent; mononuclears, 1 per cent. This patient was very difficult to manage; she would not remain quiet and constantly tugged at her bandage, complaining that it disarranged her hair. She would not co-operate with the nurses. Posterior wound closed all except at one point at about the center. This discharge persisted. Acriviolet 1,200 was introduced through the fistula on several occasions but failed to stop the mucous discharge. The principal organism identified at the time of the mastoidectomy, as well as that taken from the fistula, showed streptococcus hemolyticus. Patient discharged April 26, 1943, 10 days after operation. May 6, 1943 — Patient reported to the clinic and examination of the wound showed the fistula reduced in size to that of a pin-point. Comment — This wound was filled with acri-violet 1-200 as a control test.

Case 7: C. T., female, age 6 years, admitted March 1, 1943. Upon exami-

nation the left ear was found to be draining profusely. Pinna of ear inflamed due to the discharge. No mastoid tenderness present. Patient was kept under observation and ear kept clean by means of dry sponging. X-ray examination showed clouding and thickening on the left side suggestive of mastoid infection. Sagging of the posterior wall was observed at no time. March 22, 1943 — Mastoidectomy, left. The incision made was that of a radical mastoid going well into the hair line. Periosteum and skin separated from bone until entire mastoid bone was exposed. The mastoid was then exenterated, the antrum entered and the wound cleansed. There was considerable purulent material around the antrum. The mastoid bone was of the infantile type and contained only a few small cells. The tip cell was unusually small. After all bleeding points were ligated, the cavity was filled with equal parts of sulfanilamide and sulfathiazole powders. Periosteum approximated with No. 1 catgut, more sulfathiazole dusted between the periosteum and skin, and skin closed with Michel clips. No drain introduced. Sterile piece of gauze was introduced into auditory canal to prevent prolapse of the posterior wall. Sterile dressing. March 2, 1943 — Hemoglobin, 86 per cent, 13.4 gm. per 100 cc.; R. B. C., 4,700,000; W. B. C., 17,700; mature neutrophiles, 77 per cent; immature, 4 per cent; eosinophiles, 4 per cent; lymphocytes, 18 per cent; mononuclear, 1 per cent. March 25, 1943 — Hemoglobin, 81 per cent, 12.6 gm. per 100 cc.; R. B. C., 4,250,000; W. B. C., 12,600; mature neutrophiles, 50 per cent; immature, 4 per cent; eosinophiles, 7 per cent; lymphocytes, 36 per cent; mononuclears, 1 per cent. March 3, 1943 — Hemoglobin, 90 per cent; 17 gm. per 100 cc. R. B. C., 4,840,000; W. B. C., 11,000; mature neutrophiles, 45 per cent; immature, 4 per cent; eosinophiles, 19 per cent; lymphocytes, 32 per cent. March 18, 1943 — Culture from ear — 24-hour — many Gram positive cocci and diplococci; 36-hour — hemolytic staph. aureus — strep. viridans — nonhemolytic strep. March 22, 1943 — Culture from wound — 12-hour — Gram positive diplococci; 48-hour — nonhemolytic strep. March 29, 1943 — Culture from wound — 24-hour — many Gram positive cocci and diplococci; three-day — staph. albus. Wound healed by primary intention. On the fifth postoperative day a small opening appeared about the middle of the wound. Upon examination hair contamination was found. Wound cleansed with alcohol and culture taken (staph. albus). Patient discharged April 6, 1943, 15 days following operation. May 6, 1943 — One month later patient was examined in the clinic and the wound was found well united; scar somewhat elevated. Ear canal absolutely dry. The child, however, showed symptoms of measles and was sent home and the board of health notified.

All of the cases reported had frequent urinalyses as well as differential blood counts made during their hospitalization. Following their discharge from the hospital, they were observed at various intervals for two months.

OBSERVATIONS.

No dressings were applied after the sutures were removed. This was done on the fifth day. Aural discharge ceased in about four days, canals remaining dry. A slight leucopenia (20 per cent) and a 10 per cent reduction in the red blood cell count along with an increased lymphocytosis were observed about the fourth or fifth day. This change corrected itself within 48 hours. There were no toxic manifestations in any of the cases. No sulfa crystals were found in the urine or

blood in any case. The youngest case was 5 months and the oldest, 49 years.*

CONCLUSIONS.

1. Rapidity of cure.
2. Lessening of hospitalization.
3. Little or no annoyance to the patient.
4. Absolutely no depression or deformity postaurally.
5. No further dressings, which fact is a blessing to both operator and patient, especially children.
6. Early return of adult to work.

BIBLIOGRAPHY.

1. Blake, Clarence J.: Results of Blood Clot Dressing in Mastoid Surgery. *Trans. Am. Otol. Soc.*, 1891.

Blake, Clarence J., and Reik, Henry Ottridge: Surgical Pathology and Treatment of Diseases of the Ear. New York and London, D. Appleton and Co., 1906, pp. 252-255.

2. Reik, Henry Ottridge: The Blood Clot Dressing in Mastoidectomy Considered Physiologically. *Jour. A. M. A.*, March 31, 1906.

3. Sprague, Frank B: The Blood Clot of Wound Repair in Aural Surgery. The Laryngoscope, Sept., 1906.

4. Bryant, William Sohier: The Modified Blood Clot in Mastoid Surgery. *Ann. Otolaryngol., Rhinol. and Laryngol.*, Sept., 1906.

5. McCullagh, Samuel: The Modified Blood Clot in Mastoid Surgery. *N. Y. Med. Jour.*, June 13, 1908.

6. Holmes, Christian R.: The Value of Blood Clot in Operations for Acute and Chronic Mastoiditis. *Lancet Clin.*, Jan. 18, 1913.

7. Davis, George Edwin: The Blood Clot Dressing with Modified Technique Which Insures Painless Healing Without Deformity. Annual Meeting of the Medical Society of the State of New York, Syracuse, May 7, 1919.

8. Coates, George Morrison: Blood Clot Dressing in Mastoid Surgery. Read before the Section on Eye, Ear, Nose and Throat Diseases of the Medical Society of the State of Pennsylvania, Pittsburgh, Oct. 5, 1920.

9. Livingston, G. S.: Local Sulfonamide Therapy in Acute Mastoiditis. *Jour. A. M. A.*, pp. 117-1080. Sept. 24, 1941.

10. Currier, Wilbur D.: Sulfathiazole in Mastoidectomy Wounds. *Ann. Otol., Rhinol. and Laryngol.*, 51:955-960, Dec., 1942.

11. Tucker, C. A., and Flake, C. G.: Sulfadiazine in Management of Simple Mastoidectomy Wounds. *N. Eng. Jour. Med.*, pp. 227-1021, Dec. 31, 1942.

12. Fenton, Ralph A.: Local Use of Sulfathiazole in Otolaryngologic Practices. *Arch. Otolaryngol.*, April, 1943.

---

*From the Department of Otorhinolaryngology of the New York Medical College, Flower and Fifth Avenue Hospitals, Dr. J. A. W. Hetrick, Director.

I gratefully acknowledge my indebtedness to Dr. David Soloway (Resident Department Otorhinolaryngology, Flower and Fifth Avenue Hospitals) for his valued assistance at these operations.

SYMPOSIUM: PROGRESSIVE DEAFNESS. A — PREVENTION AND ALLEVIATION OF PROGRESSIVE DEAFNESS.*

EDMUND PRINCE FOWLER, M.D., New York.

In this paper I am giving merely an outline of my own methods of procedure and my philosophy.

Progressive deafness is an inadequate diagnostic term because progressive deafness may be due to almost any of the disorders and diseases directly or indirectly attacking the ear if they are sufficiently severe, recurrent or prolonged.

When is deafness progressive and when is it not progressive? This is a simple question but the answer is not always simple. It is a matter of timing. I think of deafness as being progressive when consistently on successive dates, over a prolonged period of time, the threshold audiograms show a change in loss of 10 dcb. or more, at two or more frequencies, whether or not some of the other frequencies temporarily show improvement. I believe that a deafness is progressive when, over a period of months or years, several persons notice that the patient cannot hear sounds that were formerly heard quite well, or if he must get closer and closer in order to hear speech clearly, particularly in large rooms or auditoriums; or if certain other auditory phenomena are observed, such as the fact that hearing for speech seems progressively better, or progressively worse, in loud environmental noise. How may we ascertain when these changes in hearing are real and when they are merely imaginary?

The most accurate means for measuring the hearing is the properly calibrated audiometer properly used, in spite of the fact that, as usually employed, the audiometer measures merely the threshold of hearing at the different frequencies and that it definitely shows only what the patient cannot or does not hear, but not necessarily what he can or does hear.

---

*Read as part of a Symposium at the Meeting of the New York Academy of Medicine, Section on Otolaryngology, April 19, 1944.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 1, 1944.

Does the audiogram correspond to the hearing for speech? The answer is "not necessarily, because in the understanding of speech many factors are involved besides the mere ability to sense a single frequency, or any group of frequencies."

Discrete tones are the simplest sounds for testing. They require less cerebral collaboration than any other frequency setup. The cortical centers are not vital to the sensing of simple sounds. Animal experiments (Culler) show that the cortical centers for speech may be removed without destroying the response for simple sounds.

When the audiograms of the right and left ears are similar, both ears are used to hear speech, but when one ear is deafened even as little as 10 dcb. below an opposite hard-of-hearing ear, the poorer ear is used comparatively little, and in many people this imposes a deafness for speech on the poorer ear out of proportion to the loss indicated in the audiograms. All of these phenomena have been taken into consideration in setting up my method of estimating the percentage of loss of hearing for speech.[1]

Some will say it does not matter what the audiogram shows, it is how one hears actual speech that matters. This is true in a way, but nevertheless the only accurate measure of capacity to hear is an audiometric measurement at the different frequencies. Note that I say "*capacity to* hear" not "ability to interpret" or "the incentive or willingness or desire to understand" speech, but simply the "capacity to hear," whether or not such capacity is used. If not used it is still available for the individual to call upon whenever he needs it and uses sufficient intensive concentration and practice to make the most use of it.

Otosclerosis is the condition usually thought of as synonymous with progressive deafness, in spite of the fact that in probably less than 12 per cent of the people harboring otosclerosis does this disorder cause deafness[2] and even in a smaller percentage continuous progressive deafness. Autopsies on otosclerotics show that about 12 per cent had ankylosis of the stapes. In uncomplicated otosclerosis it is the ankylosing process which is mainly accountable for the deafness. In the general adult population the percentage of otosclerosis is about 5 per cent, but as one-twelfth of 5 per cent is about

0.4 per cent, probably less than 0.5 per cent of the general population suffer deafness from ankylosis due to otosclerosis. Deafness from other sources is, therefore, about 10 to15 times this figure.

Nerve (perception) deafness, and particularly the nerve deafness coincident with old age or that caused by acoustic trauma, is also commonly considered as synonymous with progressive deafness. This also is not necessarily true, because even in old age and after long exposures to sound traumas, in many instances nerve deafness from many causes, even though it is severe and permanent, is not progressive; at least not rapidly enough to be placed in the category of progressive nerve deafness.

Conduction deafness, due to the effects of nonsuppurative or suppurative lesions in the middle ear and its contiguous cavities, if prolonged or recurrent, may cause progressive deafness; but it too is not synonymous with this term, because in many instances the hearing remains about the same for many years.[2]

It would, therefore, seem obvious that we should abandon the term "progressive deafness" as an entity, unless we qualify it by one or more of its underlying causes. To do this would be scientific and would aid in treatment, because it would focus the attention more upon the etiology than upon the symptoms. It would lead to more careful examinations and intelligent treatment.

Faulty surgery is one cause of progressive deafness. It may be too long delayed, too conservative, meddlesome or too radical. As in other categories of medicine and surgery, the first consideration is, and properly so, the saving of life; but operators should be not only good surgeons, but good otologists as well. They should not allow patients to drift insofar as function is concerned. There are many reasons for neglecting threatening hearing defects; a major one is that there may be sufficient hearing to get along with, especially if there is a better hearing capacity on the opposite side. One good ear is sufficient for very good hearing under most environmental noise conditions.

There are signs that otologists are emerging from this ancient custom of *laissez faire* and beginning to appreciate how the preservation of hearing depends not alone upon how the ear looks, or how trivial a congestion or thickening appears to the naked eye, but also upon how the ear functions under the lesions remaining after the threatening inflammatory or degenerative episodes have subsided.

The ear should not be treated as an isolated unit in the body economy, because it may be affected by diseases existing in almost any tissue and even by many psychosomatic upsets which in the past have been considered too often as merely hysteric episodes. It seems that these conditions have been considered as having nothing to do with the hearing, whereas as a matter of fact they may and often do have not only an immediate influence, but also, if sufficiently severe and repeated, they may have a permanent and progressively depressive influence on the hearing. This subject is very important, but I merely mention it here in passing.

The praiseworthy endeavor to hasten the healing of wound cavities, to obtain a primary wound closure, is questionable practice in instances where the middle ear drainage will be insufficient, without additional drainage by way of the additus ad antrum. Everyone acknowledges that if in a suppurative otitis media the perforation in the drum prematurely narrows or closes it threatens an exacerbation or prolongation of the suppuration and indicates that incisions or other operative procedures to re-establish drainage should not be delayed. Premature narrowing or closure of the mastoid wound is also fraught with similar hazards. Prolonged ear suppurations are always a threat to the hearing. Of course, if on cleaning out the mastoid cells it is seen that there has been no inflammatory destruction of the tissues, in other words, if the case is one that would probably have recovered without operation, then go ahead and concentrate on quick closure of the wound, but not otherwise.

I am convinced that the deafness following many mastoidectomies could have been prevented if two things had been kept in mind and appropriate action taken: *1.* Do not delay operating after you have decided that there is danger of prolonged suppuration in the middle ear or mastoid, and

especially if there have been prior recurrent middle ear suppurative episodes; *2.* if there is any question of a delayed resolution, establish or re-establish and maintain adequate drainage of the suppurating cavities by manipulations or operative procedures.

The properly timed and executed simple and modified radical mastoid operations are important measures for preventing and alleviating progressive deafness from acute or prolonged suppurative middle ear, attic, antrum or mastoid involvements. Both are often postponed or avoided until too late to prevent deafness. Childhood is the period of best results, and the removal of hypertrophied lymphoid tissue from the nasopharynx a most practical aid. If the lymphoid hypertrophies persist after surgical adenoidectomy, irradiation is good preventive and curative treatment.

Careful and repeated tests for function of the neural mechanism of hearing and for the physiological functioning of the conducting mechanism is always indicated. How can this be done? The prime requisites are careful audiometric examinations of the hearing and pneumometric examinations of the Eustachian tubes and middle ears. (Note, I use the pleurals.) Both ears should be protected, not only the ear immediately concerned, but the opposite ear as well.

The hearing tests are now so well known that I will not discuss them at this time. Let us rather devote a few words to the test for patency of the Eustachian tubes. In my opinion the best test for physiological tubal patency is to have the patient perform Toynbee's experiment, or swallow while he maintains a negative pressure in the nasopharynx and then with a magnifying speculum observe whether or not the drum retracts during these procedures, and particularly if it returns to its former position after the patient subsequently swallows. If it does, the tube is certainly patent and it is probably physiologically patent, because it has opened under adverse circumstances; if it does not it is not physiologically patent. The same observation holds true for automatic tubal ventilation by swallowing during rapid barometric variations in airplanes, elevators and tunnels.

We should disabuse ourselves of the persistent notion that mere patency to catheterization necessarily means a normally

functioning tube, and that consequently there exists a normal ventilation of the middle ear and surrounding spaces. On the other hand, in some ears, although neither the patient nor the examiner can hear air entering the middle ear, otoscopic observation may reveal that it does so with facility. In such instances it seems obvious that treatment by catheterization is superfluous and usually ill advised.

One-tenth of a drop of mucus in the Eustachian tube is enough to interfere with its physiological functioning, even though it permits some ventilation of the middle ear from time to time. In this connection it should be noted that when mucus secretion is blown into the middle ear it may not be detectable by auscultation. To remove the threat of deafness there must be no obstruction in the tube when it is insufflated.

What is the best way to remove excessive secretion and prevent its recurrence? I have found that regular gentle Politzerizations in the office, supplemented at least three or four times a day at home by my little balloon inflator, in over 90 per cent of cases will produce a favorable result and greatly diminish the number of office visits. Sometimes irradiation is necessary, and if properly used it will definitely aid in diminishing the mucus in the nasopharynx and tubes and in resolving lymphoid hypertrophies. Bouginage even with the radium bougie should be used with reluctance and great caution.

The radical mastoid operation is in disrepute as a means for improving the hearing because if the loss of hearing is less than 45 dcb. it is usually made worse by the operation. The radical operation is now almost entirely reserved for chronic suppurative conditions which threaten serious complications; however, if some way can be found to prevent postoperative excessive scar tissue from forming over the labyrinth windows I am sure that the radical procedure could be employed in many instances to improve the hearing. It may be that improved techniques will some day provide such a means. I have seen instances of remarkable improvement in the hearing after radical operations, even in those revealing extensive cholesteatoma and a large fistula in the external semicircular canal. In one instance, although the fistula closed,

I observed a hearing loss in the most important speech frequencies of only 15 to 25 dcb. This is good hearing for close speech, and requires the use of no artificial hearing aid. I have seen such improvements exist off and on for several years, even though the fistula closed. In some cases upper respiratory episodes dulled the hearing but on their passing the hearing returned to its former level. At times a slight pressure upon the tissue over the labyrinthine windows was necessary to re-establish good hearing. What does this mean? It seems to mean that we do not know just how the ear functions under pathologic conditions; it means, perhaps, that the presence of a patent fistula is not always so important as we have been led to believe; it means that much research is necessary before we find ways and means to improve the hearing in cases where we cannot cure the lesions causing the deafness, where we cannot bring the ear structures back to their predeafness perfection.

No discussion on the alleviation of deafness would be complete without mentioning the fistulization operations, which are now front page news, and which have engendered much acrimonious and emotional controversy. This much may be said in all fairness: Information is being obtained. Recent reports from those doing the great majority of these operations show some astounding results and instances wherein the hearing has remained at what is called "a satisfactory level" for five or even six years. If these favorable results are substantiated, it would be justifiable to allow certain patients to risk the fistula operation. Of course, they must be fully informed of its present status, of the risks involved, the postoperative reactions and the necessity of follow-up and that in no given instance can any surgeon surely predict a satisfactory or lasting improvement in the hearing.

Strange to say, in many instances after fistulization of the labyrinth, although the hearing is not improved, the patient seems to hear more, perhaps because he pays better attention and tries to hear, and to read the lips. He really then is psychologically benefited although there may be little or no change in his hearing. In such cases other measures could have produced similar results. I believe the fistula operation ulti-

mately may be of service not only in otosclerosis but also in other forms of obstructive deafness. Let us hope so.

The symptom most often associated with deafness is tinnitus. It is often neglected as a warning signal and treated as a symptom of importance only for its nuisance value. The cause of tinnitus should be sought and if possible controlled. It usually can be found and controlled to a large extent, often completely.

It might be that we are on the threshold of epoch-making accomplishments for the relief of deafness; but no matter what such corrective measures may accomplish, successful prevention will accomplish more. It is usually difficult to institute and enforce preventive measures, but this should be our ideal and goal. Not for one moment should we allow ourselves to neglect preventive treatment nor such alleviating measures as hearing aids and other means toward rehabilitation. The societies for the hard-of-hearing have taught us much along these lines. The cooperation of all otologists in this work is needed if we are to increase the faculties toward the pursuit of happiness of those who are threatened with diminishing hearing.

REFERENCES.

1. Fowler, E. P.: A Simple Method for Measuring the Percentage of Capacity for Hearing Speech. Fundamental Factors in Setting Up a Standard. *Arch. Otol.*, 36:6:874-890, Dec., 1942.

2. Guild, Stacy R.: Personal Communication.

3. Fowler, E. P.: Hearing Reclamation and Preservation in the Moderately Deafened Child. *N. Y. St. Jour. Med.*, 35:7:287-294, April 1, 1935.

4. Fowler, E. P.: Physiology of the Eustachian Tube. *Jour. A. M. A.*, 1909.

140 East 54th Street.

AMERICAN BOARD OF OTOLARYNGOLOGY.

The third edition of the Directory of Medical Specialists listing names and biographic data of all men certified by the 15 American Boards is to be published early in 1945. Diplomates are requested to make prompt return of the notices regarding their biographies as soon as possible after receiving the proper forms soon to be mailed from the publications office.

SYMPOSIUM: PROGRESSIVE DEAFNESS. B — PRESENT STATUS OF MEDICAL AND SURGICAL THERAPY FOR THE DEAFENED.*†

SAMUEL J. KOPETZKY, M.D., New York.

Therapy for the deafened is still for the most part an empirical procedure. This is as true of surgical treatment, based upon diagnosis and indications predicated upon the present state of our knowledge as it is of medical therapy. We do not know exactly how either accomplishes its results.

Exact diagnosis of the lesions causing progressive deafness is possible only in a small number of patients. We realize that we are dealing mostly with numerous and varying conditions, all of which present one symptom in common — varying degrees of deafness.

Serial audiometric studies of bone conduction, under conditions of masking, often show acoustic dysfunction for a few weeks, with a return of function after such an interval of time. There is no satisfactory explanation for this. Diagnosis, based on examinations made during the period of dysfunction, is often wrong because the findings change as the loss of function becomes restored and a different diagnosis is in order.

Most otologists are surgeons; therefore, the surgical approach to the problems of the deafened has been emphasized. Surgery is based upon the assumption that, if sound waves could be brought to the acoustic nerve end-organ the problem would be solved. This is an over-simplification of the problem and the surgical approach has failed in approximately 50 per cent of cases. A further reason for failure is that evidence is lacking in the literature that any progress has been made in separating those cases in which surgery has a reasonable chance of resulting in permanent practical improvement of hearing from those in which such a hope is vain.

*Read as part of a Symposium at the Meeting of the New York Academy of Medicine, Section on Otolaryngology, April 19, 1944.

†From the Department of Otolaryngology, New York Polyclinic Hospital and Medical School.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 1, 1944.

We have as yet no way to separate cases almost identical in respect to hearing loss, age, sex, prior medical history, and otoscopic and physical findings, in some of which operation results in an appreciable gain in hearing; in others, no permanent practical improvement is obtained.

The acceptance of a surgical operation by the patient still entails *a gamble* on his part as to whether under the most auspicious circumstances, the operation will bring the desired improvement in hearing.

The solution of the problem of surgical therapy lies not in small improvements of details of technique incidental to a more modern surgical procedure based upon the pioneer work of Holmgren and Sourdille, but in improvement *in differential diagnosis*. I venture to hazard the guess that the first step in such differential diagnosis will rest on an improved comprehension of the physiology of hearing and a better understanding of the rôle which all parts of the auditory apparatus play in the function of hearing.

Crowe, seeking explanation for the loss of high note frequencies among the aging and old, called attention to minute fractures of the small bony ridges radiating from the semicircular canals. He contended that these breaks in continuity of bone was a reason for the failure of sound waves of high frequency to reach the end-organ in the cochlea.

Whether this is true or false is beside the question; however, it denotes *thought* toward ascertaining reasons for the hearing loss which clinicians find upon examining individuals over 50 years of age.

In preparing myself to perform the fenestration operation, in order to have a membranous labyrinth which was fresh and not dried, shriveled and disintegrated, I procured very recently deceased cadavers. In a few, I noted a tense membrane spanning the antrum, its attachment in front lost in the tissues of Prussak's space about the incus, and attached and lost behind in the lining membrane of the additus ad antrum. Unfortunately, I made no record of it, believing it an artifact. In a few of the patients upon whom I performed the fenestration operation, I again saw this membrane. Again I failed to include it in my notes of operative findings.

Recently, hunting through old anatomical textbooks, I found it illustrated in Sobotta's Human Anatomy.

What is the function of this tense membrane? Can it be a vibrating membrane having to do with the function of hearing?

Obviously any degree of infection would very easily destroy this membrane, and its absence would thereafter be a factor in the hearing loss. At present this is only a presumption on my part. The fact that surgeons have never noted it in operative work on mastoid infections supports the theory of its easy destruction by infections of the middle ear spaces.

When the problem of differential diagnosis is solved — and I do not profess to know the answer at the present time — then a more exact selection of cases for surgery will result; and, incidentally, it will be possible to judge more exactly the results obtained by various surgeons who are pioneering in this field.

I still believe that there is a difference in cases which is *not shown* by the medical history, by the X-rays of the mastoid, by tuning fork tests or audiometric examinations. The points of differential diagnosis emphasized by Holmgren, namely, an atrophic skin of the external auditory canal wall, rather large external auditory canal, an atrophic membrana tympani with a slow or absent vascular reflex, a lessened or absent cough reflex and a freer mobility of the posterior-superior quadrant when under observation during pneumatic suction and compression have not proved adequate. I have records of observations on hundreds of cases regarding these factors and have found that they do not furnish the required differential diagnostic data.

In cases with a positive heredity, I usually found prolonged bone conduction over air conduction and a raised lower tone limit.

Regarding serial audiometric tests: I do not consider one or two otoscopic examinations and a few audiometric and fork tests sufficient to furnish more than casual observation. When serial audiometric tests are made, in the course of a period of some months' study, then a better picture of what is hap-

pening to the hearing acuity during this period becomes apparent. The effect of intercurrent conditions such as allergies, common colds and other incidents show upon the record.

Time does not permit a discussion of the effects of allergy, or of cardiovascular disease and kidney dysfunction with their concomitant body water imbalance. All these deleteriously affect hearing acuity and as they become superimposed upon the basic lesion which deafened the patient, the deafness will be increased.

Therapy for the deafened must include therapy directed toward relief or cure of these concurrent conditions.

There is one other item that must not be overlooked. All otologists have commented on the fact that in chronic progressive deafness (otosclerosis) the significant lesion in the bony labyrinthine capsule presents periods of quiescence during which hearing loss is stationary. During these periods when the hearing level remains stationary, the accompanying tinnitus sometimes becomes less intense. In studying the serial audiometric findings, it is obvious that any conclusion must take into account that during a period of quiescence the lessened masking effect of the tinnitus results in improved hearing. This must be discounted in judging the effects of therapy. In other words, one should not ascribe improved hearing to therapy but should recognize it as incidental to one of the intervals of inactivity of the disease. To judge whether the disease is in such a period of latency requires considerable diagnostic acumen.

SURGICAL THERAPY.

The basic foundations for surgical therapy were developed by Holmgren and Sourdille. Everything that has been added is but a modification of their pioneer work. The essence of surgical therapy is the fenestration of the horizontal semicircular canal, the maintenance of patency of the fenestra. The methods of reaching the operative field, whether postaurically or endaurally, and the manner of creating and adapting the flap to cover the window are matters which vary in the hands of different surgeons. In some cases I have removed the incus; in others, I have not, with equally good results. The results of surgery have been the subject of many

reports, and the summary given by Dr. Marvin Jones to the American Academy of Ophthalmology and Otolaryngology last October is perhaps the most recent authoritative resumé. The chance of success is approximately 50 per cent. The definition of what constitutes a success still needs clarification. By and large, one may ascribe success to the procedure, if permanently improved hearing of practical value results from the surgery. As I stated in my introductory remarks, I am unable to predict whether or not a given case will be a success, even if every procedure and step in the surgery is carried out correctly. It is still the patient's gamble.

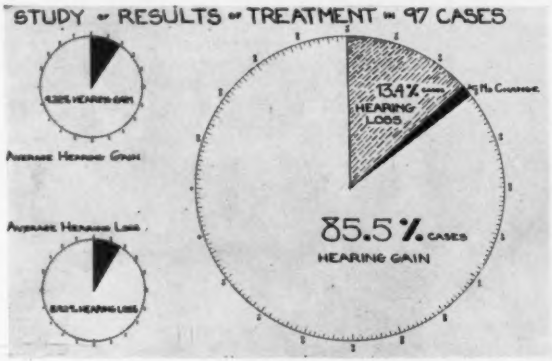

Chart 1. In Chart 1 is shown diagramatically a summation of the 97 cases; 85.5 per cent demonstrated a gain in hearing, 13.4 per cent did not gain, but demonstrated a further loss of hearing, and about 1 per cent plus remained stationary.

The average hearing gain was 8.92 per cent and the average hearing loss during the period of treatment was 9.22 per cent.

I present the audiometric studies of three cases, which have been published elsewhere,[1,2] as sufficient to substantiate the contentions about surgery made in this discussion. These cases have been under observation for from three to four years. The fenestra has remained open, the ears are dry, and the hearing results have remained good. One was operated upon, following the technique of Sourdille, the other two were fenestrated after employing the postauricular route of approach. In one of these the incus was removed, in the other it was not; however, in both cases the head of the malleus was removed.

What must be realized by both patient and surgeon is the fact that *normal hearing is not obtained by fenestration of the semicircular canals.* Only a practical gain is attainable, providing the loss of hearing has not fallen below the 40 dcb. level prior to operation.

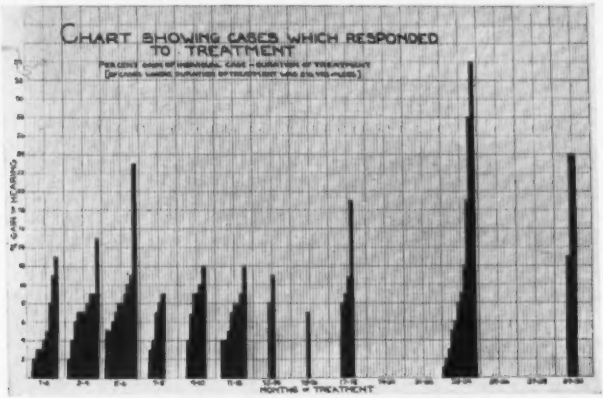

Chart 2 shows the gains studied against the months under treatment. Each square on the chart represents one case. The largest numbers show appreciable gains in the first six months of treatment. One case showed its greatest gain after two years of treatment.

No case should be subjected to surgery until it has been under observation and medical treatment has been tried for a considerable length of time, preferably for at least six months. In addition there shall have been made detailed studies regarding the patient's general condition. I do not advise any surgeon to operate upon cases referred to him unless he has a detailed record showing that all known medical therapy has been tried for a considerable length of time without result, and unless there is a detailed record of hearing level deterioration over a period of time, which distinctly demonstrates a continuing and progressive hearing loss. I suggest that each surgeon make his own personal observation over a time period long enough to enable him to know definitely that the hearing loss is actually and persistently increasing.

I have seen too many patients who have been told that unless they submitted to surgery they would become totally deaf within a short time. Some of these have their hearing acuity recorded in the graphs submitted herein, and what

they informed me they were told was finally proven to have been untrue.

Finally, before surgery is suggested to the patient, a properly fitted hearing aid should be used for a period of time, the results of its use as to social and business adjustments awaited. The very use of a hearing aid has therapeutic value.

Surgery, in my opinion, should be reserved for those cases which present an evident rapid deterioration of hearing *in spite* of medical treatment and for cases where hearing impairment is below the level of practical hearing; namely, about 40 dcb. loss (average) and no improvement is obtainable by medical means.

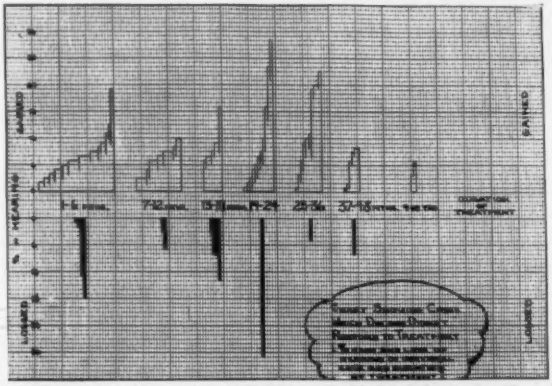

Chart 3 shows hearing gains and losses studied against months of treatment. Again it is evident that both the greatest gains and the losses can be estimated during the first six months of treatment.

MEDICAL THERAPY.

In the first place in those cases where deafness is very severe, including those with congenital deafness, it is necessary to separate those who have psychosomatic dysfunction from those whose deafness is due to an organic lesion. It must be remembered that all deafened persons present a degree of psychoneurotic involvement. There is no time in this presentation to detail the reasons and the types of *sensory word aphasia* often mistaken for deafness. The separation of those with psychogenic type of deafness is possible by employing the Lowenstein examining apparatus and not-

ing the psychosomatic reactions of the patient. This will be reported later when the research work is resumed after the war.

The medical treatment must consist in handling allergies and of water imbalance by suitable medication and diets.

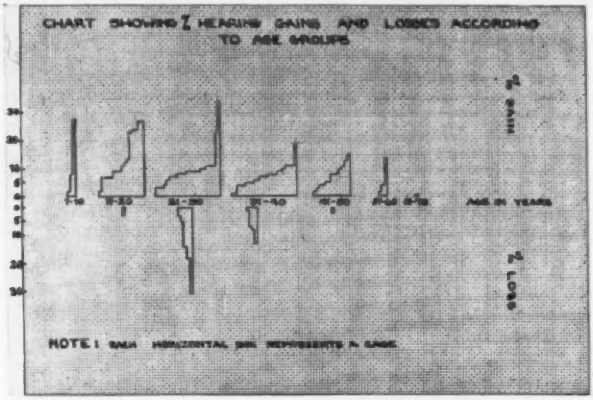

Chart 4 represents a breakdown of these 97 cases into a graphical demonstration of the hearing gains and losses already seen in Charts 1 and 2, but in Chart 4 studied against the age group concerned.

In children, particularly, Crowe has made a very valuable contribution. Children whose lymphatic tissue about the mouth of the Eustachian tube is hypertrophic, and who show hearing loss, can have their hearing acuity restored by using Crowe's method of radium application in the area of the pharyngeal orifice of the Eustachian tube.

Instead of introducing radium, I have employed radium emanations, planned so that a period of application of from nine to 10 hours gives a dosage of 250 millicurie hours of radiation. My results have been very satisfactory, and fully substantiate Crowe's contentions.

At a recent meeting of the Section on Otolaryngology of the New York Academy of Medicine, Dr. Jacob Jacobson, of my staff, presented his work on the use of benzyl cinnamate injections to control tinnitus and thus improve hearing also. This has been so recent that I shall not refer further to it.

For a long time, otologists have held to the idea that a richer blood supply to the middle and internal ear spaces would give beneficial results, by causing reabsorption of inflammatory infiltrations, and also tend to relax tension in the tensor tympani and stapedius muscles, when these hold the ossicular chain immobile by being in spastic simultaneous contractions.

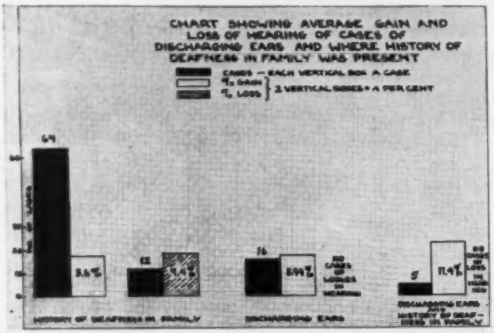

Chart 5 shows a breakdown of the 97 cases, showing a hearing gain of 8.6 per cent in 64 cases in which there was an absence of any history of discharging ears at any time, and where there was a positive heredity of history of deafness in the family. Of such cases there were 12 which lost hearing while under the vitamin therapy.

We have begun to study the audiometric records before and immediately after injection of the *stellate ganglion* with novocaine. This produces a homolateral dilatation of the blood vessels. One can observe the hemorrhagic injection of the membrana tympani a few minutes after the injection. This study is temporarily suspended "for the duration." Nevertheless, to make this presentation complete, I must make note of it. In the cases so far subjected to this procedure no bad effects have thus far been noted. I shall report upon this measure in full at some future time. The technique used at our clinic was developed by Dr. Ned Shnayerson.

DRUGS.

Of all the drugs and biochemicals which I have tried, the administration of the precursor of Vital A (Carotine-in-oil; Caritol) in doses of 25,000 units a day, with yeast concentrate (B complex) in doses of 4 gr., given three times a day, has given me the best results.

Additionally, once a month, I give these patients a subcutaneous injection of anterior pituitary extract (alopecia formula) of 1 cc.

The anterior pituitary extract must be avoided during the menstrual periods. Dietary habits, the interdictions of the use of mineral oils in food salads or as purges is necessary.

Dr. M. Joseph Lobel, of the Polyclinic Staff, made a record of more than 300 cases of hearing loss in patients in whom he demonstrated a vitamin deficiency. The administration of the needed vitamins had a profoundly beneficial effect upon the hearing. His study is as yet not completed. It will be reported subsequently.

I herewith present my personal study of 97 cases in which vitamin therapy was used. Space prohibits the inclusion here of the details of each of these cases. The summaries of these cases is presented in the accompanying graphs.

Included among these 97 cases are some having or having had suppurating ears. Some, in my opinion, have otosclerosis, others have a progressive deafness which is as yet undifferentiated. No vitamin deficiency studies were made on these 97 cases.

In Chart 1 there is shown diagramatically a summation of the 97 cases; 85.5 per cent demonstrated a gain in hearing, 13.4 per cent did not gain but demonstrated a further loss of hearing, and about 1 per cent plus remained stationary.

The average hearing gain was 8.92 per cent and the average hearing loss during the period of treatment was 9.22 per cent.

Chart 2 shows the gains studied against the months under treatment. Each square on the chart represents one case. The largest numbers show appreciable gains in the first six months of treatment. One case showed its greatest gain after two years of treatment.

Chart 3 shows hearing gains and losses studied against months of treatment. Again it is evident that both the greatest gains and the losses can be estimated during the first six months of treatment.

Chart 4 represents a breakdown of these 97 cases into a graphical demonstration of the hearing gains and losses already seen in Charts 1 and 2, but in Chart 4 studied against the age group concerned.

Chart 5 shows a breakdown of the 97 cases showing a hearing gain of 8.6 per cent in 64 cases in which there was an absence of any history of discharging ears at any time, and where there was a positive history of deafness in the family. Of such cases there were 12 which lost hearing while under the vitamin therapy.

There were 16 cases which had discharging ears when they first came under observation. During the course of treatment no further hearing loss was observed; on the contrary, these 16 gave an average of 8.94 per cent of hearing gain.

There were five cases which had discharging ears when they came under observation and in addition gave a history of familial deafness. Of these, under vitamin treatment there was an average gain of 11.4 per cent and no further loss of hearing was demonstrable during the period of observation.

Conclusions: It is obvious from the study of these charts that the hearing may be held at its existing point when we are dealing with chronically discharging ears which do present indications for surgery. The hearing may be held level by vitamin therapy. This also seems true in cases with family history of deafness. This type of medication may also result in hearing improvement.

Finally, if I compare the conventional hearing charts of patients treated medically with those treated surgically, there is little difference in the total of hearing gained. Thus one may conclude that a period of observation, study and medical therapy should always be tried before suggesting surgery to the patient as a means to improve his hearing. Personally, I reserve surgical therapy for those cases which fail to respond to any known nonsurgical therapy, and in which while under observation there is very definite recorded evidence of progressive loss in hearing acuity.

It must be clearly understood when surgery is advised that, at the present stage of our knowledge, the results cannot be predicted.

Finally, the use of hearing aids should be advocated more generally. They play a useful part in therapy and they enable the patient to hear.

REFERENCES.

1. KOPETZKY, S. J.: Studies in Labyrinthine Fenestration to Improve Hearing (Preliminary Report). THE LARYNGOSCOPE, Nov., 1939.

2. KOPETZKY, S. J.: History and Present Status of Operations on the Labyrinthine Capsule for Otosclerosis. *Surg., Gynec. and Obst.*, 72:466-489, Feb. 15, 1941.

SECOND SOUTH AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

The second South American Congress of Otorhinolaryngology will take place in Montevideo during the first half of October, 1944. The following speakers have been designated:

Argentina: Prof. Eliseo V. Segura, Collaborator: Prof. Eduardo Casterán. Subject: Prophylaxis of Deafness.

Bolivia: Prof. Felix Veintemillas. Subject: The Postoperative Care of Mastoiditis.

Brazil: Dr. Plinio de Mattos Barretto. Subject: Bronchoscopy as a Diagnostic and Treatment.

Chile: Dr. Riesco, M.C. Subject: The Importance of the Examination of the VIIIth Nerve in Tumors of the Brain.

Paraguay: Dr. Crispin Insaurralde. Subject: Leishmaniosis in Otorhinolaryngology.

Peru: Prof. Juvenal Denegri. Subject: Rhinoscleroma.

Uruguay: Prof. Justo M. Alonso. Subject: Cancer of the Larynx.

The official speeches and the communications must be in the hands of the General Secretary of the organizing committee before July 31 and Aug. 31, respectively.

The fee is $12—Uruguayan pesos—and those who wish to participate should address the President of the Sociedad de Otorhinolaryngology of his country, or the General Secretary of the Executive Committee, Dr. Juan Carlos Oreggia, Yi 1491, Montevideo, Uruguay.

SYMPOSIUM: PROGRESSIVE DEAFNESS. C — REHABILITATION.*

WESTLEY M. HUNT, M.D., New York.

Rehabilitation is a long word with broad meaning. Seeking to narrow it within the boundaries of otological discussion, I have tried to define it. What is rehabilitation? What does the word mean, in terms of progressive deafness, which is the subject of our meeting here tonight? The dictionary definition of "rehabilitate" is "to restore to former capacity; reinstate," and from that I have arrived at a definition of my own with which I believe doctors who have had wide experience with hard-of-hearing patients will agree. It is this: Rehabilitation is the process of restoring an individual to himself.

Progressive deafness makes deep inroads on the personality; it attacks the dignity; it undermines self-confidence; endangers security, psychological as well as financial. Rehabilitation of hard-of-hearing, then, must be largely concerned with intangibles, with the restoration of invaded dignity, self-confidence, security — with the restoring to the individual of his heritage as a human being. It is the purpose of this paper to discuss the otologist's share in this restoring process, both in terms of individual patients and of the hard-of-hearing in general. We consider here only persons suffering from progressive deafness, which, though its roots usually reach back to childhood, tends to escape serious attention until early adult life. Rehabilitation of the congenitally deaf, who must be specially educated and trained, is considered a separate problem, which is being admirably handled by well qualified state and private institutions.

What, then, is the otologist's part in the rehabilitation of the hard-of-hearing? I can tell you where it begins: It begins in the office, with the individual hard-of-hearing

*Read as part of a Symposium at the Meeting of the New York Academy of Medicine, Section on Otolaryngology, April 19, 1944.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 1, 1944.

patient. From there it extends outward into the fields of medical science, research, psychiatry, public health, education, social service, legislation. I cannot tell you where it ends.

Let us begin in the otologist's office with a typical hard-of-hearing patient. The fact that the patient has arrived at the otologist's office means two things: First that he has reached the point where he can no longer conceal his handicap, even from himself; second, that he is worried about his job.

Looking at this man, the otologist can readily visualize the boy he used to be. The boy who had the bad case of measles at the age of eight, perhaps. The one who was picked on in school for inattention, jeered at for making mistakes; who had to be forced to take part in group activities; who wasn't interested in girls. Making his painful way through childhood and adolescence, unguided, unaware of why he was always at a disadvantage, the young man chose his career. Salesmanship, probably, or teaching, or some other profession in which good hearing is essential. It is well known to psychologists that the handicapped, if unguided, tend to choose occupations which specifically require the ability in which they are deficient.

Now the man is grown. In spite of his unacknowledged handicap, he has forced from life the things he most wanted — a good job, marriage, family. He has arrived at the otologist's office — why? Because he has everything the normal man wants out of life and he is in mortal fear of losing every bit of it.

The otologist who examines this patient and writes on his record card, "Progressive deafness," has made only superficial diagnosis. The record might more accurately read: "Diagnosis: fear."

Fear of failure, fear of ridicule, fear of people; fear of new situations, chance encounters, sudden noises, imagined sounds; fear of being slighted, avoided, made conspicuous — these are but a handful of the fears that haunt the waking, and even the sleeping hours of the sufferer from progressive

deafness. Small wonder that, at best, he tends to live in an atmosphere of despondency and suspicion. Small wonder that, at worst, he may not particularly want to live at all.

Otologists know these things but they are inclined to forget them, perhaps because, having good hearing themselves, they tend, as all normally-hearing people do, to underestimate its importance. I am not suggesting that otologists plug their ears one day a month in order to understand their patients' problems better, though that might not be a bad idea. What I do suggest, however, is that otologists realize that they have fulfilled their function only when, in addition to giving all possible medical care, they have seen their patients well started on the road to rehabilitation.

It is taken for granted that the otologist, after examining the patient suffering from progressive deafness, is going to tell him the truth; but there are many ways of telling the truth. Incredible as it may seem, there are still first-rate otologists who talk to hard-of-hearing patients as if they were children — children of low-grade intelligence, at that; who assure them cheerfully that there are worse things than deafness. "Suppose you were going blind"; who remark that hearing is a matter of "paying attention" and dismiss the patient's distracting head noises as "probably imaginary"; who wave the whole difficulty away with, "Look at Thomas Edison. He said he was lucky to be deaf; he didn't have to listen to a lot of nonsense!"

When otologists talk that way, is it any wonder that hard-of-hearing patients flock by thousands to quacks and miracle men who take their problem seriously, treat them like adults, and, so long as they are solvent, promise to "restore their hearing completely without fail?"

Since the patient is emotionally disturbed, and the otologist is likely to be the first and sometimes the only person with whom he frankly discusses his problem, it is hardly sufficient merely to tell him the truth about his hearing and give him necessary medical care. The otologist must assume the rôle of guide, philosopher and friend; he must explain and open up avenues to the compensatory forces which exist for this particular handicap.

Chief among these, of course, are lip-reading and hearing aids. The former is considered so essential that a program of intensive lip-reading instruction has been set up in three specially designated Army hospitals for returning war-deafened veterans. Yet there are still first-rate otologists who have only a sketchy idea of what lip-reading is, how and where it is taught in their localities, what benefits can be expected from it.

Hearing aids have so vastly improved during recent years and have put so many of the hard-of-hearing back into employment that the War Production Board has ruled their manufacture to be essential in wartime; yet there are still first-rate otologists who have only a sketchy idea of what hearing aids are, how and where they are selected, what benefits may be expected from them.

The otologist must know, not only what his patient needs for rehabilitation, but also where he can get it. He should know where he can receive lip-reading instruction; where he can test and compare the different hearing aids. He should be ready to refer him for vocational guidance and retraining, if necessary, or for psychiatric help, if this is indicated. Also, the otologist has definite responsibility to preach deafness prevention to this patient, knowing that he might have avoided progressive deafness if he had had proper medical care after that attack of measles at the age of eight, and that the patient has children of his own.

In New York there are certain rehabilitation facilities for all the acoustically handicapped with which every otologist should be familiar. If he were familiar with only one of these, however, it would be sufficient, since this one, in addition to rendering its own rehabilitation services, is a reliable storehouse of information on all other facilities available. I refer to the New York League for the Hard-of-Hearing, to which an otologist can refer a patient for expert advice and consultation on hearing aids, lip-reading, vocational guidance and hearing problems in general. At the League the patient can get not only specific help but also the mental lift and reassurance which come from association with League staff members who are themselves hard-of-hearing and have achieved successful rehabilitation.

Other facilities available are the New York State Bureau of Rehabilitation; the Veterans' Administration and Army hospitals for war-deafened veterans; private and public schools for lip-reading instruction; Centers in Universities, such as the Department on Exceptional Children at Teachers College, Columbia University, which has a division on the deaf and hard-of-hearing; special committees on the hard-of-hearing which exist from time to time in the National Research Council; committees of the New York State Medical Society, the County Medical Societies and the Division on Otolaryngology in the American Academy of Ophthalmology and Otolaryngology.

So much for the otologist's responsibility in the rehabilitation of the individual patient. We come now to his responsibility to the hard-of-hearing in general — a far-reaching responsibility, since the whole subject of rehabilitation, in the wartime present and looking ahead to the postwar future, is opening up before our eyes, disclosing new and hitherto undreamed-of possibilities. It is up to otologists to be leaders, not taggers-along, in the field of rehabilitation of the hard-of-hearing.

All such rehabilitation programs, whether local, state or national, will benefit immeasurably by active cooperation from otologists. Community programs in schools, hospital clinics and Leagues for the Hard-of-Hearing need leadership from otologists, who, both as individuals and through their medical society affiliations, can do much to obtain necessary legislation for putting such programs into effect.

Otologists should familiarize themselves with labor laws, compensation laws, rehabilitation laws concerning the physically handicapped and work toward their improvement. They should know what are the measures for checking the exploitation of the hard-of-hearing by advertisements of fake cures, treatments and appliances, and if state measures are not effective, otologists should make them so.

Otologists should be active in public education, taking all opportunities, by public addresses or radio broadcasts, to talk on deafness prevention, conservation of hearing and aural hygiene in general. They should spread the gospel of deafness

prevention to doctors of medicine, who need more otological information. They should try to interest psychiatrists in the particular problems of the hard-of-hearing and work more closely with them.

Ours is a responsibility, as I have said, which has no end. Today there are millions of men who, every day, risk potential deafness — in active duty on the fighting fronts, in defense factories on the home front. We have no way of knowing how many hearing casualties there will be; we simply know there will be thousands and that we must be here, ready to restore them to themselves and, in a wider sense, restore them to the world as valuable and contributing members of society. Only if otologists know what rehabilitation is, know what facilities are available and work for better ones — only then can we feel confident that the deafened may come to us and find what they need.

907 Fifth Avenue.

AN UNUSUAL CASE OF OSTEOMA OF THE NASAL ACCESSORY SINUSES.*

JAMES S. HANLEY, M.D., New York.

Osteomas of the nasal accessory sinuses while not rare are not common. I feel that they are considered rare due to the fact that, as in my own experience, they have not been reported. They are true bony benign tumors of two distinct types: *1.* the solid ivory-like structure which is most common and in X-ray appearance is denser than the bones of the skull; *2.* the cancellous, which is often mistaken by X-ray for a chondroma. The solid type is attached to the skull either by a broad or a pedunculated base and as a rule may be removed in one piece, although the one with the broad base is much more difficult to remove and keep intact. One might think that the cancellous type was the structure of a bony type in its earlier development, but this is not true. Observation of these osteomas over a period of years shows that they never change in structure. If they start as the bony type or as the cancellous type, they remain bony or cancellous.

As to etiology, symptoms and pathology, I cannot add anything which has not been covered so excellently by Johnston in his article, "Osteoma of Frontal Sinus."

There are many of these cases which are seen in routine Roentgen examination of the skull and nasal accessory sinuses which would never have been diagnosed, except as an incidental finding, as the three main symptoms of headache, dizziness or deformity were not complained of. Until the tumor does give symptoms which may be attributed to it, there is no necessity for intervention as they grow very slowly and may never cause the patient any trouble. The type of operation for the removal of the tumor depends upon its situation, its size and extent of damage done to the surrounding structures by pressure.

---

*Read at the Meeting of the New York Academy of Medicine, Section on Otolaryngology, New York, Nov. 17, 1943.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Dec. 6, 1943.

REPORT OF CASE.

F. K., white male, aged 17, was first seen in the Eye Clinic at the New York Eye and Ear Infirmary, Feb. 20, 1943. His chief complaint was that the left eye seemed smaller than the right eye.

History revealed that this patient was struck in the left eye by a handball six to eight months previously. Approximately one month following this accident the patient noticed that the left eye was somewhat smaller than the right one and was pushed back. He also began to notice recurring frontal headaches of about one hour's duration. He had noticed the swelling of the inner canthus of the right eye for the past four years. There were no complaints of visual disturbance.

The left eye showed an enophthalmia. The pupils were equal and reacted well, no diplopia, near point 175 mm. (normal 75 mm.), media clear, fundi-discs swollen and veins slightly engorged. Vision 20/30 both eyes.

Physical examination and routine laboratory tests were all normal. The Kahn and Klein tests were negative.

Roentgen Examination (Dr. Irving Schwartz): A large, dense, calcified mass occupies almost completely the frontal sinuses and the right half of the ethmoidal labyrinth. The mass in the frontal sinuses measures 7 cm. in breadth, 4 cm. in height and 3 cm. in depth at its thickest part. The tongue-like projection extending into the right ethmoidal region measures 3 cm. in height, 1.5 cm. in width and 3 cm. in anteroposterior diameter. The right ethmoidal mass bulges into the right orbit and to a lesser extent into the mass in the right frontal region. The mass in the left frontal sinus bulges into the left orbit. The mass is probably an osteochondroma. There is no X-ray evidence of fracture of the nasal bones.

The patient was referred to my clinic for examination and operation. Examination showed a hard, fixed, palpable, non-tender tumor above the inner canthus of the right eye. Ears and throat were negative. The nose was negative except for some hypertrophy of the right inferior turbinate. There was no paranasal sinus tenderness.

Operation: A butterfly incision was made just above, and not through, the eyebrows and carried across the bridge of the nose down to bone. The skin and periosteum were reflected upwards. There was a circular dehiscence of the outer table of the left frontal sinus, about 1.5 cm. in diameter, with the tumor mass protruding into it. The anterior wall of both frontal sinuses and the supraorbital ridges were of eggshell thinness and were removed easily with a curette. The left frontal sinus except for a narrow groove at its most superior border was completely filled with a dense ivory-like mass. The same condition was found in the right frontal sinus except the mass was cancellous. The floor of the left frontal sinus was practicaly all gone and the mass occupied part of the orbit. The tumor in the left frontal sinus was attached in the region of the nasofrontal duct and was removed in toto. The cancellous tumor was removed with a curette and had a broad attachment to the posterior wall of the right frontal sinus and had also removed part of the floor. After removal of the cancellous tumor on the right side, a third mass of ivory-like consistency was seen protruding into the most medial part of the sinus. This tumor extended downwards, occupying the ethmoidal labyrinth, and outwards, eroding the ethmoidal plate. It occupied part of the orbit. This mass was also removed in toto and had a pedunculated attachment in the region of the right nasofrontal duct. Neither supraobital

ridge could be saved due to its thinness. The skin edges were approximated and a cigarette drain was led from each frontal sinus into the nasal cavity. The wound healed by primary intention and considering the extent of the tumor masses the patient has comparatively little deformity. Following the operation, the patient had a diplopia which disappeared in six days. This was due to disturbance of the trochlea of the superior oblique muscle. His headache and deformity of the eye have not returned.

The reasons for reporting this case are:

1. Enophthalmus previous to operation, exophthalmus being usually present in these cases.

2. The multiplicity of the osteomas in the same patient. There were three distinct and separate tumors.

3. The two different types of osteomas, ivory-like and cancellous, in the same case.

40 West 55th Street.

ALLERGY IN RELATION TO OTOLARYNGOLOGY AND OPHTHALMOLOGY. A REVIEW OF THE RECENT CURRENT LITERATURE.

FRENCH K. HANSEL, M.D., St. Louis.

During the past year comparatively few articles in the literature have been directed to the subject of allergy as related to otolaryngology and ophthalmology. In general, the articles which have been selected for review represent distinct advances in this field of medicine.

PHYSIOLOGY AND PATHOLOGY.

Fowler[1] reports an interesting case of a woman 34 years of age who developed a typical Horner syndrome on the left side following removal of the left stellate ganglion for the relief of pain in the left arm. Obstruction and profuse watery discharge appeared on the left side of the nose. Smears of the nasal secretion from this side showed eosinophiles. Skin tests were negative. The symptoms were controlled with Syntropan. Among four other patients with Horner's syndrome, one had symptoms similar to the above patient. It was not stated whether eosinophiles were demonstrated in the nasal mucus in this case.

Mohun[2] reported a group of eight cases in which nasal blocking or congestion developed during pregnancy in women who had never had such symptoms when not pregnant. Five of these women had had similar symptoms during one or more previous pregnancies. The symptoms disappeared within one to seven days after delivery.

In another group of 12 women who already had hay fever or nasal allergy, the symptoms were markedly increased during pregnancy. Mohun concludes that the increase in estrogen during pregnancy is probably the etiologic factor.

Unilateral polyposis not infrequently occurs in older children and adolescents. dos Reis and Corrêa[3] report their obser-

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 27, 1944.

vations on a group of seven cases. The patients varied in age from 12 to 21 years, most of them being in adolescence. The Caldwell-Luc operation was successfully performed on all of them. This type of polyp is usually presented in the choana, the attachment being most frequently in the antrum. It is important to differentiate this type of polyp from that of allergic origin. The choanal polyp is not the result of allergy; therefore, it should not be erroneously treated as such.

A most outstanding contribution has been made by Hilding[4] on the relation of ciliary insufficiency to death from asthma and other respiratory diseases. He points out that there is a group of cases, among patients who die from asthma, which shows a very striking and characteristic change in the bronchial epithelium. The change consists of a substitution of goblet-like cells for the normal columnar ciliated cells. Apparently it is a true metamorphosis. As this change takes place, the ciliary mechanism is lost and the characteristic viscid, mucinous secretion accumulates in the air passages. Normally this viscid secretion is very readily carried up vertical surfaces by ciliary action. The amount of secretion is enhanced because the erstwhile ciliary cells are secreting as well as the glands. The difficulty of removal of the secretion is aggravated because the mucin remains attached over large areas within the cells which produce it, thus anchoring the mass to the wall. When the air passages have become completely filled, the patient dies of asphyxia. In Hilding's opinion, this metamorphosis is the chief pathologic change and death results directly from the loss of ciliary function.

He noted a second group of asthmatics in which the cilia were also lost, but the picture was essentially different. This group was characterized by chronic bronchitis with purulent secretion. There was destruction in the surface of the bronchial epithelium and the ciliated cells had sloughed off very extensively. In these patients also the air passages filled up with secretion to such an extent that the patient died. Attention is also called to the fact that bronchospasm was doubtless a factor in some of these cases. In one of the group which was omitted from this discussion, spasm seemed to play a dominant rôle. A patient with tracheobronchitis who died in

extreme respiratory distress showed the ciliated epithelium practically entirely destroyed, and all the bronchi contained viscid plugs of secretion. Further studies were conducted on pathologic material from the pandemic of influenza in 1918. The material was not entirely satisfactory, but as far as could be determined the bronchiolar epithelium had been entirely destroyed and all the cilia with it.

In all of these groups, Hilding feels that the mechanical removal of the secretion as a substitute for ciliary action is indicated. Aspiration, either through a bronchoscope or a tracheotomy, would be the best.

In a series of animal experiments, Hilding[5] has shown that certain pathologic conditions in the upper and lower respiratory tracts, such as postoperative pulmonary atelectasis, vacuum headache and retraction of the eardrum, are the result of insufficiency of ciliary activity.

The steps in the development of postoperative pulmonary atelectasis were outlined as follows: "*1*. An excess of secretion is formed within the affected lobe. *2*. A succession of occluding masses, or pistons, of mucus form across the lumina of the air passages. *3*. These "pistons" move up the cylindrical air passages by ciliary action, each carrying a quantity of air. *4*. As soon as the pressure within the lobe begins to fall, it shrinks by its own elasticity and from pressure by adjacent lobes. *5*. The adjacent lobes carried by the force of inspired air move into the space relinquished by the affected one. *6*. The advancing pistons rupture serially as they reach tubes of greater diameter and meet more forceful changes of air pressure. Each then releases the bubble of air which it carried and continues on its course as a mural mass of film. *7*. A negative pressure of considerable proportion is produced within the lobe when the supply of air is exhausted. *8*. The masses of secretion then present in the air passages come to a standstill when the cilia can no longer advance them against atmospheric pressure. *9*. The cilia continue to remove secretion from these stalled masses in thin films and might eventually remove them entirely if they were not replaced by continued secretion."

In the experiments regarding vacuum headache, he noted

that negative pressure can be produced within a normal sinus by the introduction of a quantity of mucus which replaces a portion of the contained air. When the ostium becomes occluded, negative pressure develops within the sinus. The pressure falls as the mucus passes through the ostium. The pressure ceases to fall when it becomes equalized on the inside and the outside. The cilia continue to act and remove the mucus in thin films. When sufficient mucus has been removed, atmospheric pressure forces air into the sinus again. In this manner, Hilding states, negative pressure almost certainly develops clinically in sinuses more or less filled with mucus.

The negative pressure which is known to occur in the middle ear is probably caused by the force of ciliary action moving mucus down the Eustachian tube.

DUSTS AND MOLDS.

In a group of 100 selected patients, Davidson[6] noted that of 95 reacting to house dust, 90 also reacted to animal hairs. In this group he felt that animal hairs were responsible for the production of symptoms in only 10 per cent. This brought up the question as to whether the animal hairs contacted in other ways than from live animals were responsible for the activity of house dusts. On the basis of clinical observation and skin tests, Davidson concludes that the final proof of the constitution of house dust awaits the development of tests by which to differentiate the almost endless number of allergens in it. Some investigators have presupposed the presence of some unknown ingredients in house dust which would account for its activity. He finally concludes that house dust is composed principally of cotton, flax, jute, wool, silk, six or more animal hairs, three or more feathers, glue, kapok, orris root, pyrethrum and tobacco.

Durham[7] has presented a very comprehensive treatise of the subject of air-borne fungus spores as allergens. His report is based upon extensive observations made in the United States, Alaska and some foreign countries. A comparative study of the total seasonal fall of alternaria and hormodendrum spores and ragweed was made in 53 cities. In number and in widespread geographic distribution, the

spores of stem rust, alternaria and hormodendrum are outstanding. The daily fluctuations of mold spore concentrations are more pronounced than those of pollen and are not as easily accounted for by weather conditions as are fluctuations in the pollen curve.

The importance of these air-borne fungus spores as a cause of allergic conjunctivitis, nasal allergy and asthma cannot be too strongly emphasized. In the central states area we have found them to be the cause of respiratory allergy during the spring, summer and early fall, in a significant number of cases. These patients may or may not be sensitive to pollen; as a rule they are not, but have been considered as pollen-sensitive by a number of observers. In most instances, however, skin reactions to pollen were considered as borderline or negative. In the diagnosis and treatment of these cases we have employed an extract of feed mill dust such as that described by Wittich.[8,9] This dust contains all the spores noted on slides exposed for making the pollen counts. Positive skin reactions to this extract have been consistent and treatment with small doses has resulted in most satisfactory relief. In a number of cases presenting a rather typical history of the vernal type of conjunctivitis, the response to mold therapy was also satisfactory.

ALLERGY AND PEDIATRICS

Clinical papers on allergy in children by Deamer,[10] Long,[11] Criep,[12] Miller[13] and Marks[14] emphasize the importance of the diagnosis and treatment of respiratory allergy. Attention is called to the fact that symptoms simulating the common cold, characterized by sneezing, nasal obstruction and discharge, are frequently overlooked as allergy and treated as infection. There should be no difficulty in making the distinction between allergy and infection if the nasal secretions are examined for the presence of eosinophiles or neutrophiles. It is important to consider the common cold, however, as a complication of a nasal allergy. During a cold there may be an absence of eosinophiles. Follow-up examinations in these cases will reveal an eosinophilia after the cold has subsided. Some of these observers call attention to the frequent unnecessary removal of tonsils and adenoids in these cases considered as

infections. Chronic persistent cough in children is often a forerunner of asthma and should be carefully investigated from the allergic standpoint.

THE NOSE AND PARANASAL SINUSES.

Shambaugh[15] reports his observations on the differentiation of infectious and allergic rhinitis. In 102 consecutive cases of chronic nasal or sinal disease which he reviewed, the condition was essentially nasal with secondary involvement of the sinuses in 51. In 43 of these there was response to allergic treatment. In the other 51 cases the disturbance was primarily sinal and in 36 of these a response to treatment for allergy was obtained. He emphasizes that both the infection and the allergic condition must be treated in order to give maximum relief.

In the management of allergy as related to otolaryngology, Hansel[16] emphasizes the importance of diagnosis of nasal and sinus allergy as well as other associated manifestations. In the treatment of hay fever small doses of pollen extracts given subcutaneously or intracutaneously are recommended. Both preseasonal and coseasonal methods may be employed. In the treatment of dust sensitives, small doses are also recommended, starting with a 1-10,000,000 dilution in small children, 1-1,000,000 in older children and in adults with severe symptoms. Most patients received satisfactory relief with maximum doses of 0.10 to 0.20 of 1-100,000 or 0.10 to 0.20 of 10,000. In only a few cases was a 1-1,000 dilution employed. On the whole, the skin tests as a means of determining the relative degree of sensitivity to dust was found unreliable. Very good results were obtained in cases in which the skin reaction was entirely negative. In general, the administration of weak or stronger doses was based upon the degree of clinical sensitivity or severity of symptoms. Weak dosage was employed in the severe cases and stronger dosage in the mild cases.

In the study of cases of so-called recurring sinusitis, bronchitis, colds or catarrh in Southern California, Smith, Goodhill and Webb[17] point out that such conditions are caused by an unrecognized or hidden pollen allergy. The pollen allergy

problem in Southern California presents great difficulties owing to perennial pollen present in the air. They state that continual exposure to perennial pollens results in the development of a low grade resistance which is not sufficient to prevent allergic symptoms but does result in minimum reactions to the usual skin tests. Perennial pollen treatment, including empirical treatment with the pollens indicated by the patient's history and environment when tests were inconclusive was found to be of definite value.

In the diagnosis of pollen allergy, the skin reaction is considered a very reliable guide. Minimal or borderline reactions always present a problem in treatment. It is important to take into consideration the possibility of atmospheric mold sensitivity in those cases. If the patient's symptoms parallel the pollen count, treat with pollen extracts; if they parallel the atmospheric mold count, treat with molds. In some instances treatment with both may be indicated. According to our experience in the central states area, therefore, most of the questionable pollen cases have proven to be mold cases.

In a group of 80 cases of chronic maxillary sinusitis in which an antrum window operation was performed, McHenry[18] reports that in 21 the infection was of dental origin and in 23 allergy was a factor. Among the allergy cases, 18 were relieved of their sinus symptoms. In the entire group of 80 cases, 60 were considered as cured, 12 were relieved of symptoms, and in eight the results were unsatisfactory.

In a personal study of 250 patients who had had operations on the nose and sinuses, Hollender[19] found that the results were satisfactory in 65 per cent of the nasal cases and in 38 per cent of the sinus cases. According to the results reported by 154 rhinologists in a reply to a questionnaire, the "cures" reported were 71 per cent for nasal surgery and 42 per cent for sinus surgery. Hollender finally concludes that the chief factors in unsuccessful operations are: Inaccurate diagnosis; neglect of allergic factor; failure to appreciate the importance of systemic diseases, such as diabetes, syphilis or tuberculosis; too hasty surgical intervention; poor surgical technique or incomplete operations; and inadequate postoperative care.

According to the observations of Urbach and Gottlieb,[20] vasomotor rhinitis was encountered in 38 per cent of 379 cases of asthma. This percentage seems far too low. In children with asthma we found the incidence of nasal allergy almost 100 per cent. In adults with asthma, the incidence of nasal allergy is about 90 per cent. These estimates were corroborated by positive cytologic findings in the nasal secretions. Nasal allergy associated with asthma is often very mild, so much so that the patient does not complain about it, yet a diagnostic eosinophilia is present.

After 14 years' study of nose and throat problems in relation to asthma, Weille[21] concludes that the rhinologist cures asthma in about 10 per cent of the cases referred to him. Symptoms are relieved in about 40 per cent for shorter or longer periods of time, and no results are obtained in the remaining 50 per cent. The cases seen by the rhinologist, he states, represent the failures of the allergist to a great extent and are, therefore, a special group. For better results, a closer collaboration between the rhinologist and the allergist and a better understanding of the relation of sinusitis to asthma is advocated.

DRUGS.

During the past few years a number of synthetic ephedrinelike drugs have been developed for the treatment of respiratory allergy. In general, an attempt has been made to eliminate those factors which cause a rise in blood pressure, palpitation and nervousness.

Friedman and Cohen,[22] in reporting the use of a new synthetic ephedrine-like drug — Nethamine — in the treatment of hay fever and asthma, state that of 23 cases of hay fever, 14 were improved and of 23 cases of asthma 11, or 48 per cent, were improved. The incidence of such symptoms as nervousness and insomnia was definitely less with nethamine than with ephedrine, while nethamine was at least equally as effective in relieving symptoms. The heart rate and blood pressure were unaffected.

Fabricant and Van Alyea[23] report their observations of the use of privine hydrochloride, a new imidiazole derivative,

2-naphthyl-methyl-imidiazoline. It is crystalline and readily soluble in water or normal saline. In animal experiments it was found that a 0.1 per cent solution had no detrimental effect on ciliary activity. In normal subjects it caused no alteration in the pH of the secretions. It caused no irritation of the mucous membrane or any of the toxic-side effects as sometimes noted with ephedrine.

Scott[24] reports two cases, both in children, in which severe reactions to ephedrine occurred, although both patients had been treated previously with ephedrine without ill effects. One patient, a girl, age 12 years, had used a nasal spray of 2 per cent ephedrine and had also taken it internally without ill effects. Later, following the use of 1 per cent ephedrine in the nose, she developed a severe reaction of chilliness, dryness of the throat, palpitation and restlessness. The second patient, a boy, age 6 years, who had hay fever and asthma, had taken ephedrine internally in doses up to $\frac{3}{8}$ gr. with $\frac{3}{4}$ gr. Amytal without ill effect. Later, during an acute infection with asthma, the same dosage was followed by a severe reaction characterized by restlessness, anxiety, thirst, rapid pulse and respiration. Scott stated that such violent reactions to ephedrine in children normally tolerant to the drug seemed inexplicable, but point out the necessity for caution in the administration of ephedrine.

Although the above reactions to ephedrine might be considered of an allergic nature, it also appears that they may simply have been the result of overdosage. A dosage of $\frac{3}{8}$ of a grain of ephedrine is very large for a child; even many adults will not tolerate it. In general, an average dose of $\frac{1}{8}$ gr. for a child and $\frac{1}{4}$ gr. for an adult is sufficient. In some instances large doses are required for effectiveness and may be well tolerated.

Girling[25] reports his observations on the use of histamine in 120 cases which included 73 cases with postnasal drip with other evidence of allergy such as intermittent nasal obstruction; 14 cases of nasal allergy with conductive deafness; 20 cases of lower half headache with nasal symptoms; six cases of asthma; five cases of dermatitis and two cases of laryngitis. Intracutaneous tests were made with 0.01 cc. of a

1-100,000 dilution of histamine diphosphate. An area of erythema over 35 mm. in diameter was considered a positive reaction. In the treatment of positive reactors, the dilutions used were: 1-100,000, 1-75,000, 1-50,000, 1-25,000 and 1,10,-000. When the first evidence of reaction appeared, the dosage used was reduced to two-thirds of the last dose; this was further reduced if reaction continued. From 25 to 40 treatments were given. After the course of treatment, the skin reactions were greatly reduced or negative. Of 73 cases of nasal allergy, 70 were completely relieved; of the 14 cases of nasal allergy with conductive deafness, five were completely and eight partially relieved; 15 of 20 lower half headache cases were entirely relieved; of six cases of asthma, five were completely relieved. Four of five cases of dermatitis were relieved. Both of the cases of laryngitis were completely relieved. Girling concludes that it remains to be determined whether these results are permanent.

Criep[26] reports the case of an allergic patient who developed nasal allergy and asthma following the topical application of arygyrol. A positive scratch reaction was noted with a 1 per cent solution. An intracutaneous tests with this solution resulted in a severe local and a mild constitutional reaction. Ophthalmic and passive transfer tests were also positive. Tests with several other preparations of mild silver protein were negative. The protein molecule associated with the argyrol was believed to be the sensitizing factor. Criep is of the opinion that argyrol may be a frequent cause of sensitization and may be the cause of the discomfort some patients experience from its use.

Kauvar and Mount[27] report their observations on a study of 127 patients with common colds, of whom 75 were treated symptomatically and 52 with sulfonamides. The usual dosage of the sulfonamides was 2 gm. initially, followed by another 2 gm. in two hours, then 1 gm. every four hours. Treatment was continued from three to eight days. No complications were noted. The sulfonamide-treated patients averaged 5.01 days and the control group 5.26 days in the hospital. From these studies it was concluded that the use of sulfonamides in the treatment of mild upper respiratory infections is not justified. Toxic reactions or sensitivity may

develop, thus possibly making it impossible to use in the case of the development of a more serious illness. They recommend the use of sulfonamides, therefore, only in the complicated cold cases.

Recent reports on the use of sulfonamide drugs locally or generally in the treatment of the common cold have been controversial as to whether or not they are effective. Davis[28] reports his observations on the treatment of 157 cases treated by ordinary symptomatic drugs and compares the results with a group of 162 in which sulfathiazole was given orally in 1 gm. initial doses followed by ½ gm. every four hours. A control group consisted of 187 men and women. Treatment was given for three days. At the end of that period 70.3 per cent of those given sulfathiazole were cured and 22.3 per cent were improved. Thirty-nine per cent of those given symptomatic treatment were cured, 35 per cent improved. Of the control group, 47 per cent were cured and 29 per cent improved. The 162 treated with sulfathiazole lost 13 days from work while in the control group of 187, 96 days were lost. There were a few reactions to sulfathiazole, such as, nausea, eight cases; headache, five cases; dizziness, five cases. Davis concludes from this study that sulfathiazole treatment of common colds in industrial workers results in an important saving of working time.

Ebert[29] reports his results in the treatment of acute rhinitis in 92 patients from the use of sulfathiazole powder insufflated into each nostril daily or every other day. Relief of symptoms was almost instant and there was no irritation. The amount of sulfathiazole powder insufflated with each treatment averaged 0.0164 gm. for each side of the nose. Each patient received an average of 3½ treatments, so the total amount of drug used was only 0.12 gm. None of the patients showed the slightest toxic effect.

Our experience in the use of sulfathiazole in the treatment of the common cold is in agreement with that of Davis and of Ebert. The treatment is more effective when used early; that is, the first or second day. It should be administered about three times a day and should not be continued more than three or four days. Continued use for longer periods

seemed to set up a great deal of irritation in some cases. Overtreatment might induce local and perhaps general sensitization. A great deal of caution should be exercised in treating allergic patients with colds because of possible sensitization. If a patient has had previous toxic or sensitization manifestations from the administration of sulfonamides, it should not be used in the nose, for even the small amounts introduced can cause toxic symptoms and allergic manifestations, especialy dermatitis.

THE EAR.

Merica[30] discusses his findings in 135 cases of vertigo due to tubal obstruction. Many of the patients had associated acute or chronic disease of the sinuses or ears. In 15 instances the condition was associated with colds. Some patients derived permanent relief from one inflation. Others had recurrence of symptoms and required repeated treatments. Some patients with Eustachian tube obstruction proved to be allergic and improved when the offending allergen was recognized and eliminated. Patients with a low metabolic rate had improved tubal patency after the administration of thyroid.

Noun[31] reports the occurrence of chronic otorrhea caused by food sensitivity in two children, age 8 and 15 years. Positive skin reactions were demonstrated with several foods by direct and transfer tests and were corroborated clinically. The otorrhea subsided following the avoidance of the offending foods. Reintroduction of the foods caused an exacerbation of the otorrhea. It was suggested that the membranes of the middle ear were the site of the allergic reactions. In both patients a perforation of the eardrum had existed from a previous otitis media. No cytologic studies were made on the discharge from the ears. Both children had nasal allergy.

Although it is possible that the middle ear may be the site of an allergic reaction, in cases of the type reported in which there is an old perforation of the drum associated with a nasal allergy, it is difficult to determine whether the secretion in the middle ear arises from this site or comes from the nose as a result of blowing. With the subsidence of the nasal

allergy the aural discharge should also subside. We have observed a number of cases of this type in which an otorrhea subsided under similar conditions. This discontinuation of nose-blowing may also result in improvement or cure.

THE EYE.

Bab[32] reports his observations on allergy of the eye in a group of 83 cases, in 41 of which there was edema of the eyelids. The etiologic agents varied greatly and included sleeping pills (phamadorm) in one case; stewed cherries in another; strawberries in two others; and mechanical irritation, such as that produced by plucking the eyebrows, squeezing the margin of the lid to straighten the lashes, and dyeing the eyebrows.

O'Brien and Allen[33] report their observations on the diagnosis and treatment of allergic kerato conjunctivitis. This condition is characterized by recurring attacks of redness and swelling of the eyelids, accompanied by itching, lacrimation and discharge. A local eosinphilia, they state, may or may not be demonstrated. In one of the cases reported, orange was the offending allergen. A patch test with orange peel was positive and the injection of orange was followed by kerato conjunctivitis. In another case butyn was the cause. In three additional cases the causes were hydrous wool fat in an ophthalmic ointment, a proprietary inhalant and a fur coat, respectively.

Morrison[34] reports a case of chronic catarrhal conjunctivitis aggravated by the application of an ointment of sodium sulfathiazole and sulfathiazole. Discontinuance of its use resulted in a remission and reapplication caused an exacerbation. The base was found to be innocuous.

Marton[35] reports four cases of severe vernal conjunctivitis caused by pollen sensitivity. None of the patients had nasal symptoms. Two patients failed to show reactions to pollen. One reacted to grass and ragweed and one to grass, hickory and faintly to ragweed. All four patients were given intensive treatment with pollen extracts with good results.

In cases of this type it is important to consider atmospheric molds as a possible factor.

Brown, Irons and Rosenthal[36] made the observation that workers in laboratories producing cultures of tubercle bacilli noticed that systemic reactions resulted when the fumes from boiling suspensions of dead tubercle bacilli were inhaled. After repeated exposures, cutaneous sensitivity to tuberculin became markedly decreased. In two cases reported, recurrent tuberculous iritis had been present for many years. In neither patient could foci of infection be found. Both reacted strongly to Mantoux tests. Both patients became free of symptoms after repeated inhalation of the fumes from boiling suspensions of tubercle bacilli. A slight recurrence of iritis once followed the direct exposure of the eyes to the fumes. Animal experiments showed that sensitized guinea pigs could be desensitized by the repeated inhalation of the fumes.

REFERENCES.

1. Fowler, E. J., Jr.: Unilateral Vasomotor Rhinitis Due to Interference with the Cervical Sympathetic System. *Arch. Otolaryngol.*, 37:710, 1943.

2. Mohun, M.: Incidence of Vasomotor Rhinitis During Pregnancy. *Arch. Otolaryngol.*, 37:699, 1943.

3. dos Reis, G., and Correa, A.: Killian's Polyp. Seven Cases. *Rev. brasil. de Oto-rhino-laryngol.*, 11:17, 1943.

4. Hilding, A. C.: Relation of Ciliary Insufficiency to Death from Asthma and Other Respiratory Diseases. *Ann. Otol., Rhinol. and Laryngol.*, 52:5, 1943.

5. Hilding, A. C.: The Rôle of Ciliary Action in Production of Pulmonary Atelectasis, Vacuum in the Paranasal Sinuses and in Otitis Media. *Ann. Otol., Rhinol. and Laryngol.*, 52:816, 1943.

6. Davidson, M. T.: The Source of the Allergic Activity of House Dust. *Jour. Allergy*, 14:244, 1943.

7. Durham, O. C.: Air-Borne Fungus Spores as Allergens. *Am. Assn. for Advancement of Sci. Publ.*, 17:32, 1941.

8. Wittich, F. W.: Further Observations on Allergy to Smuts. *Lancet*, 59:382, 1939.

9. Wittich, F. W.: The Nature of the Various Mill Dust Antigens. *Lancet*, 60:418, 1940.

10. Deamer, W. C.: Some Comments on Asthma and Allergic Rhinitis in Childhood. *Univ. Cal. Med School Clin.*, 1:655, 1942.

11. Long, A. E.: Nasal Allergy in Children. *W. Va. Med. Jour.*, 39:14, 1943.

12. Criep, L. H.: The Importance of Allergy in the Practice of Pediatrics. *Pa. Med. Jour.*, 46:816, 1943.

13. Miller, B. N.: Some Problems of Allergy in Childhood. *Jour. S. Car. Med. Assn.*, 39:175, 1943.

14. Marks, M. B.: Cough in the Allergic Child. *Arch. Ped.*, 59:697, 1942.

15. Shambaugh, G. E.: Nasal Allergy. *Jour. Mich. Med. Soc.*, 42:441, 1943.

16. Hansel, F. K.: Principles of Diagnosis and Treatment of Allergy as Related to Otolaryngology. The Laryngoscope, 53:260, 1943.

17. Smith, H. D.; Goodhill, V., and Webb, M. E.: Nasal Allergy. The Otolaryngologists' Problem; in Relation to Southern California Districts. *Cal. and West. Med.*, 58:275, 1943.

18. McHenry, L. C.: Some Observations on the Maxillary Sinus. *South. Med. Jour.*, 36:18, 1943.

19. Hollender, A. R.: Nasal and Sinus Surgery. *Eye, Ear, Nose and Throat Mon.*, 22:21, 1943.

20. Urbach, E., and Gottlieb, P. M.: The Relation of Vasomotor Rhinitis to Bronchial Asthma. *Arch. Ped.*, 59:382, 1942.

21. Weille, F. L.: Fourteen Years of Practical Experience with Sinus Problems in an Allergy Clinic. *Ann. Otol., Rhinol. and Laryngol.*, 51:830, 1942.

22. Friedman, A. J., and Cohen, A. E.: Use of New Ephedrine-Like Drug in Hay Fever and Asthma. *N.W. Med.*, 42:138, 1943.

23. Fabricant, N. D., and Van Alyea, O. E.: A Note on the Evaluation of Privine as a Nasal Vasoconstrictor. *Am. Jour. Med. Sci.*, 205:122, 1943.

24. Scott, R. A. M.: Reactions to Ephedrine. *Brit. Med. Jour.*, 1:414, 1943.

25. Girling, W. N. M.: Administration of Histamine in Allergic Conditions. *N. W. Med.*, 42:196, 1943.

26. Crier, L. H.: Allergy to Argyrol. *Jour. A. M. A.*, 121:421, 1943.

27. Kauvar, A. J., and Mount, F. R.: Effect of Sulfonamide Therapy on the Common Cold. *Jour. Kas. Med. Soc.*, 44:290, 1943.

28. Davis, H. J.: Treatment of the Common Cold. *Indus. Med.*, 12:426, 1943.

29. Ebert, E.: Local Treatment of Acute Rhinitis with Sulfathiazole. *Arch. Otolaryngol.*, 38:324, 1943.

30. Merica, F. W.: Vertigo Due to Obstruction of the Eustachian Tubes. *Jour. A. M. A.*, 118:1282, 1942.

31. Noun, L. J.: Chronic Otorrhea Due to Food Sensitivity. *Jour. Allergy*, 14:82, 1942.

32. Bab, W.: Allergy of the Eye. *Am. Jour. Ophthalmol.*, 24:759, 1941.

33. O'Brien, C. W., and Allen, J. H.: Allergic Kerato Conjunctivitis. *Arch. Ophthalmol.*, 29:600, 1943.

34. Morrison, W. H.: Allergic Conjunctivitis and Dermatitis from the Topical Use of Sodium Sulfathiazole and Sulfathiazole. *Am. Jour. Ophthalmol.*, 25:1104, 1942.

35. Marton, S.: Vernal Conjunctivitis (Spring of Vernal Catarrh). *Ann. Allergy*, 1:39, 1943.

36. Brown, E. V. L.; Irons, E. E., and Rosenthal, S. R.: Results of Desensitization in Tuberculous Iritis. *Arch. Ophthalmol.*, 28:1028, 1942.

BOOK REVIEW.

Tuberculosis of the Ear, Nose and Throat, Including the Larynx, the Trachea and the Bronchi. By Mervin C. Myerson, M.D. Two hundred ninety-one pages with index and 88 illustrations. Springfield, Ill.: Charles C. Thomes, 1944. Price $5.50 postpaid.

This is an excellent book on tuberculosis of the ear, nose and throat. The author has had an enviable experience with this disease and has stated clearly and simply all the pertinent facts. The major portion of the book is devoted to tuberculosis of the larynx and is a very complete review.

The chapter on "Tuberculosis of the Trachea and Bronchus" is very well worth the attention of all endoscopists and physicians. The bibliography is complete and adequate, and the book is well prepared and well presented. It seems to the reviewer a pity that some of the illustrations fall so far short of the excellence of the text.

This book is highly recommended to all otolaryngologists, and much of it should be required reading for undergraduate medical students.

T. E. W.

SUMMER COURSE IN AUDIOMETRY AND THE FITTING OF HEARING AIDS.

STATE UNIVERSITY OF IOWA.

The State University of Iowa will feature in its 1944 Summer Session, in addition to the regular program of graduate study leading to the M.A. and Ph.D. degrees in speech pathology or hearing conservation, an intensive four-weeks course in Audiometry and the Fitting of Hearing Aids, from June 26 to July 22. This will be a practical course for physicians, physicians' assistants, public health nurses, rehabilitation aides, speech correctionists and hearing conservation specialists in both civilian and military programs. There will be three hours daily of practical laboratory work in the Otologic Clinic of the University Hospital, supplemented by three hours of lectures on the various aspects of hearing conservation, including lip reading and speech training. The course will be under the direction of Dean M. Lierle, M.D., Head of the Department of Oral Surgery and Otolaryngology, and Chairman of the Committee on the Conservation of Hearing. He will be assisted by Scott Reger, Ph.D., and Loraine Anson, M.A. Supplementary instruction will be presented by the Speech Clinic staff, Wendell Johnson, Ph.D., Director; Charles R. Strother, Ph.D; Grant Fairbanks, Ph.D., and Jacqueline Keaster, M.A.

Running concurrently with this intensive course will be a Conference Series on Speech and Hearing Rehabilitation each week-end from June 23 to July 22. The Conferences will be conducted by the following visiting speakers: Bryng Bryngelson, Ph.D., University of Minnesota; Harold Westlake, Ph.D., Pennsylvania State Department of Education; Herbert Koepp-Baker, Ph.D., Pennsylvania State College; Raymond Carhart, Ph.D., Northwestern University; Walter Hughson, M.D., Abington, Pennsylvania Memorial Hospital. These speakers will discuss the various phases of speech and hearing rehabilitation in both its civilian and military aspects.

The Summer Speech Clinic for school children and adults will run from June 19 to July 28.

CONTENTS